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THE MANAGEMENT OF COMPLICATIONS OF PEPTIC ULCER.[1]

By F. AVERY JONES, M.D., F.R.C.P.,

Physician, Central Middlesex Hospital; Consultant in Gastroenterology at the Postgraduate Medical School of London, to Saint Mark's Hospital, and to the Royal Navy, London.

I WISH to thank the Congress for the great honour of inviting me to open the discussion. I am particularly fortunate and happy to find myself in partnership with Sir Gordon Gordon-Taylor, who has done so much pioneer work on the surgical aspects of this field.

In England peptic ulcer accounts for 10% of the beds occupied by adults in the general hospitals, and therefore constitutes a major hospital problem. A big proportion of the patients are admitted with complications. In my allotted time I shall not attempt to cover all the complications, but shall concentrate on acute perforation and bleeding.

Three sections of this medical congress are meeting here to discuss the management of the complications of the peptic ulcer, and this reflects the cooperation which is needed in the medical care of these patients. I have been particularly fortunate in having close collaboration with my surgical and radiological colleagues, and my comments today reflect our joint experiences at the Central Middlesex Hospital. The great majority of those admitted to hospital with bleeding peptic ulcers, and a high proportion of those with acute perforation, have been admitted to my medical ward, and I have been responsible for their post-operative care.

[1] Read at a combined meeting of the Section of Medicine, the Section of Surgery and the Section of Radiology and Radiotherapy, Australasian Medical Congress (British Medical Association), Eighth Session, Melbourne, August, 1952.

ACUTE PERFORATION.

Until ten years ago, in acute perforation the operative mortality was commonly at least 20%, but today the figures have been reduced by at least two-thirds. Since 1938 651 patients have been admitted to the Central Middlesex Hospital with acute perforation; our experience has shown a reduction in mortality from 25% to 7%, and when patients treated surgically over the past five years are considered the mortality rate has been only 3%. It is unfortunately true that deaths still occur from delayed admission of moribund patients with peripheral circulatory failure, and of patients whose diagnosis has not been established. This may happen when patients are already very ill from some other cause. This striking fall in mortality has been due to the elimination of deaths in the first few days after admission to hospital from post-operative shock, peritonitis and pneumonia, and has been due to the routine use of antibiotics, and to improved anæsthesia with muscle relaxants.

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At the Central Middlesex Hospital there have been 651 admissions for acute perforation between 1939 and 1951 and 354 in the period from 1947 to 1951. During these five years the results have been as shown in Table I.

It is clear that there is still scope for reduction in mortality. Among the surgically treated patients deaths have occurred from induction of anæsthesia (one), post-operative shock (two), chest infections (two), intestinal obstruction (one), pulmonary embolus (one), and hæmorrhage from the ulcer (three).

Elimination of deaths from associated bleeding offers one field for further endeavour in the reduction of mortality, for associated bleeding is the main cause of death in at least a third of the present-day deaths after acute perforation. It has often been said that bleeding ulcers do not perforate, and perforating ulcers do not bleed. This is not true, at least in relation to an episode at perforation. Since 1938, 75 of the 651 ulcer patients admitted to the hospital have had associated frank bleeding. These cases fall into two groups: (i) perforation may occur with and after bleeding; (ii) bleeding may occur after perforation.

TABLE I.

Detail of Management.	Number of Patients.	Deaths.
Surgical treatment:		
Simple	316	10 (3%)
Immediate partial gastrectomy	19	0
Medical treatment:		
Perforation undiagnosed	4	4
Patient moribund on admission	9	9
Medical treatment carried out	5	3
Operation refused	1	0
Total	354	26

TABLE II.
Perforation During and After Bleeding.

Treatment Given.	1938 to 1946.		1947 to 1951.	
	Number of Cases.	Deaths.	Number of Cases.	Deaths.
Medical treatment	—	—	2	2
Surgical closure	13	6	8	1
Partial gastrectomy	—	—	5	0

Perforation During and After Bleeding.

Thirty-five of the patients in the main series developed a perforation within a fortnight of a preceding hæmatemesis or melæna. Most commonly the bleeding has occurred within the preceding twenty-four hours; in seven cases the perforation was actually unrecognized until after death. In patients with severe bleeding, particularly if they are anoxæmic, the classical signs and symptoms of acute perforation may be absent. This possibility must be kept in mind, particularly when the patient's condition seems more serious than might be expected from the bleeding.

The results of treatment in the 28 diagnosed cases was as shown in Table II. The very high mortality rate in the earlier period will be noticed, death being particularly due to repeated bleeding. It is thought that the immediate partial gastrectomy has contributed to the fall in mortality in the later period.

Bleeding After Perforation.

Forty patients have had frank bleeding at some time, often between the third and tenth days during their subsequent stay in hospital. In one case in the earlier series and in two in the latter period, partial gastrectomy was performed after bleeding had begun.

Comment.

The association of bleeding and perforation usually indicates either a double ulcer, one bleeding and one perforating, or a giant ulcer. With both possibilities radical surgical treatment is indicated, and the evidence of this series suggests that partial gastrectomy is indicated in the management of patients with this double complication.

TABLE III.
Bleeding After Perforation.

Treatment Given.	1938 to 1946.		1947 to 1951.	
	Number of Cases.	Deaths.	Number of Cases.	Deaths.
Medical treatment	—	—	1	1
Surgical closure	28	13	11	2

Prognosis.

As knowledge of the follow-up prognosis is required to assess the best immediate surgical treatment, our experience has been reviewed. Previous important studies, such as those of Illingworth, Scott and Jamieson (1946), have provided an admirable over-all picture, but such reports have not distinguished between gastric and duodenal perforations, and a limited study has therefore been made to determine whether the late prognosis is materially affected by the site of perforation. During the period from 1938 to 1948, 74 patients diagnosed as having perforated gastric ulcers were treated by surgical closure of the perforation and survived the immediate post-operative period. The subsequent course of these patients was compared with that of the same number of patients who survived the same operation for a perforated duodenal

TABLE IV.
Late Results Following Surgical Closure for Perforation of Gastric and Duodenal Ulcer.

Results.	After 2 Years.		At Follow-up, After 2 to 12 Years.	
	Gastric Ulcer.	Duodenal Ulcer.	Gastric Ulcer.	Duodenal Ulcer.
Patient untraced	4	4	6	4
Patient dead; death attributed to:				
Peptic ulcer	2	1	3	4
Carcinoma of stomach	6	0	8	0
Other disease	2	3	6	8
Total	10	4	17	12
Patient living:				
(*a*) Readmitted for gastric symptoms:				
Partial gastrectomy	9	6	15	11
Other gastric operations	3	3	3	7
Medical treatment	8	7	6	6
(*b*) Not readmitted for gastric symptoms:				
Recurrence of ulcer pain	13	18	11	14
Occasional mild pain and dyspepsia	7	6	5	5
No symptoms	19	22	11	15
(*c*) State not known	1	4	—	—
Total living	60	66	51	58

ulcer. The duodenal ulcer patients were chosen to be of the same sex and of the same ten-year age group, and to have had the perforation as close as possible before or after the corresponding gastric ulcer patients.

It will be noted that there is an important difference in the prognosis of gastric ulcer and that of duodenal ulcer, in that patients with perforated gastric ulcer have an appreciable chance of dying of carcinoma of the stomach (11% against 0%). Although there was no evidence of malignancy from the peritoneal surface at operation, nevertheless it is extremely probable that the perforations were of a primary gastric carcinomatous ulcer (Doll, 1950). Another disturbing feature is the generally poor prognosis associated with both gastric and duodenal ulcer; of the 138 patients followed up, 29 died, and of these seven (5% of all patients) died of their ulcers. The proportion who died from peptic ulcer is far higher than would be expected in a normal population, and an additional 5% mortality within an average of six years is an important consideration in the assessment of the effects of treatment, when the immediate mortality is as low as it is now. Of the

TABLE V.
Late Prognosis Among Survivors from Perforated Gastric Ulcers.

Characteristics of Patient.	Further Gastric Operation During Follow-up Period.	Test of Significance of Difference.
Sex:		
Male	27 (30%)	Not significant.
Female	9 (50%)	
Age:		
Under 45 years	17 (33%)	Not significant.
45 years and over	19 (33%)	
Length of history:		
One year or less	7 (18%)	Significant.
More than one year	29 (41%)	
Previous diagnosis of an ulcer:		
Yes	21 (50%)	Significant.
No	15 (22%)	

109 patients known to survive at the time of the follow-up examination, 26 (24%) have been subjected to a further gastric operation, and these have been specially divided in various ways to see whether it is possible to define simply the class of patient likely to require further operation.

It can be concluded that symptoms which had lasted for more than a year, or which had been severe enough to result in the diagnosing of an ulcer before the perforation occurred, are indications that the course of the ulcer disease will be unsatisfactory if the perforation is treated by simple closure.

Immediate Partial Gastrectomy.

To improve the immediate and late prognosis we have thought that there was scope for immediate gastrectomy in a proportion of cases. This was undertaken at the beginning of 1948; up to the end of 1951, 19 patients had been so treated without a fatality, and in the first six months in 1952 this number had been increased to 30. The total is too small for detailed comparison of those concerned with patients treated by surgical closure, but it would at least appear that the immediate prognosis is so good as to justify continuing with this treatment. The patients were to some extent selected to exclude "bad risks"—for example, no patient was aged more than sixty-five years; but on the other hand five patients were specifically treated by this method because of the dangerous association of hæmorrhage or perforation. Nuboer (1951) reported 65 patients treated since January, 1947, with a fatality rate of 62%, 62 having been treated by immediate partial gastrectomy. Lowdon (1952) also reported a number of patients with the same fatality rate, 51 having been treated by immediate partial gastrectomy. Altogether Nuboer and Lowden reported 113 patients treated by partial gastrectomy with one death, and our experience adds another 30 patients without a fatality. As most ulcers which perforate actually are on the anterior wall of the duodenum, the operation is technically easier to perform as an emergency for perforation than for bleeding, as bleeding ulcers are often posterior and penetrating. When the general condition of the patient is reasonably good, we suggest that this is the method of choice for a surgeon experienced in the technique when the following conditions are fulfilled: (*a*) The perforation is gastric, in the antrum or body of the stomach, and although it may appear simple there is in fact a considerable risk that the ulcer may hide an unrecognized carcinoma (the juxtapyloric ulcers have been classified under duodenal ulcers). (*b*) The patient gives a history of more than one year's dyspepsia, and an ulcer has been diagnosed previously. (*c*) Perforation and hæmorrhage from an ulcer occur concomitantly.

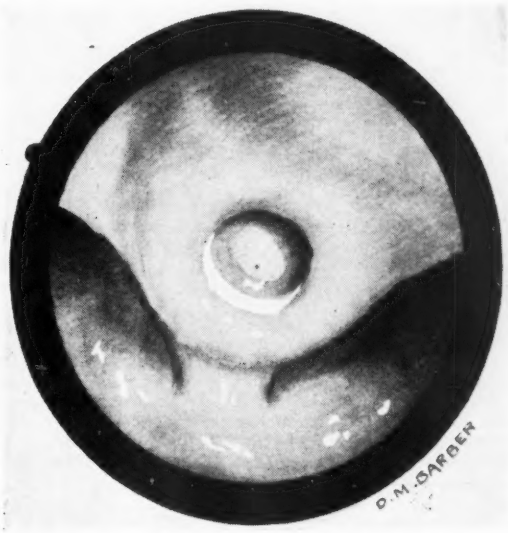

FIGURE I.
Gastroscopic drawing showing a chronic gastric ulcer.

We did not think it likely that immediate partial gastrectomy would prove to be the method of choice for all perforated ulcers, and certainly it cannot be justified in the treatment of patients in whom a duodenal ulcer has never previously been diagnosed; for such patients simple surgical closure remains the treatment of election.

Medical Treatment.

Hermon Taylor (1951) reported 73 patients treated since September, 1946, with the fatality rate of 9·6%, 65 having been treated medically.

Medical treatment has not been reported to provide a notably better immediate prognosis than the standard surgical treatment; it does not permit patients to be selected for immediate partial gastrectomy after inspection of the ulcer; there is no reduction in the incidence of lung complications (Beattie, 1951), thrombophlebitis is not uncommon (Stead, 1951), and the incidence of intraperitoneal abscesses and subphrenic abscesses is raised. There is no reason to suppose that the late prognosis is any better than that achieved by simple surgical closure. The method is contraindicated in the presence of bleeding or pyloric stenosis, for a perforation after heavy eating or drinking, and in air-swallowers. Tanner (1951) sums up as follows:

> The non-operative treatment demands frequently repeated expert clinical and radiological assessment, and any error in gastric aspiration, any momentary neglect,

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any lack of skilful observation, may be followed by disaster for the patient. . . . It is only safe in the hands of the enthusiast with ample medical and nursing assistance and is not suitable for routine usage.

The method has been shown to be practicable and is preferable under the following conditions: (*a*) When efficient surgical treatment is not readily available. (*b*) When other associated diseases—for example, recent coronary thrombosis—greatly increase the risk of surgery.

TABLE VI.
Central Middlesex Hospital, 1940 to 1950; Hæmatemesis and Melæna with Proved and Probable Peptic Ulcer.

Group.	Number.	Mortality Rate.
Chronic gastric ulcer	231	19%
Duodenal ulcer	379	8%
Post-operative	67	6%
Acute lesion	370	2%
Unclassified (mainly incomplete investigation)	65	—
	1,112	
Other causes:		
Gastric tumours, simple	4	
Gastric tumours, malignant	28	
Portal hypertension	44	
Miscellaneous	33	
Grand total	1,221	

(*c*) When perforation has occurred at gastroscopy, when the stomach is empty and experience has shown that the perforation is often no longer detectable by the time laparotomy is performed. Thus, in any one series of admission for acute perforations, it is suggested that all three methods of management—medical, simple closure, and partial gastrectomy—will have a proper place if the best immediate and remote results are to be obtained.

TABLE VII.
Central Middlesex Hospital, 1945 to 1950.

	Number of Patients.	Deaths.
No recurrent bleeding before or after admission to hospital	317	4 (1·3%)
Recurrent bleeding before, not after, admission to hospital	181	3 (1·7%)
Recurrent bleeding after (but not before) admission to hospital	100	20 (20·0%)
Recurrent bleeding both before and after admission to hospital	84	15 (17·9%)

BLEEDING PEPTIC ULCER.

The aim of treatment in bleeding peptic ulcer is to reduce the immediate mortality rate, and at the same time to make any possible contribution to the future progress of the patients. It is essential therefore to be able to visualize the factors contributing to a serious prognosis.

The important factors determining the prognosis of bleeding peptic ulcer are: (*a*) the type and site of the ulcer; (*b*) the persistence of bleeding after the patient's admission to hospital; (*c*) the age of the patient; (*d*) the presence of medical complications.

The highest mortality is among the patients with a penetrating gastric ulcer, and the lowest mortality is among those with acute gastro-duodenal lesions which are essentially superficial, and which, although they may cause very severe bleeding, nevertheless except in the elderly respond to medical management.

Naturally the recurrence of bleeding after the patient's admission to hospital is an important consideration, and its effect on the overall mortality in the peptic ulcer group is shown in Table VII.

The age of the patient is another important consideration, and in all series there is a sharp increase in mortality in the older age groups, and a low mortality under the age of forty years.

The experience from three published papers is recorded in Table VIII.

Lewin and Truelove (1949) and Baker (1947) have published mass hospital statistics from Oxford and Birmingham respectively, and these are compared with an individual series of the Central Middlesex Hospital (Avery Jones, 1947). It is true to say that in the serious emergencies of hospital work the best results come from interested individuals, and there is an argument here for relating different emergencies to different teams, or alternatively for agreeing on a common plan of action.

The presence of associated medical disease—for example, coronary ischæmia, emphysema, chronic bronchitis, calcareous aortic stenosis, or obesity—naturally increases the mortality, each partly by its own separate contribution,

TABLE VIII.
Mortality and Age.

Age (Years).	Oxford. (252 Cases.)	Birmingham. (576 Cases.)	Central Middlesex. (615 Cases.)
Under 30	0	3·0%	2·5%
30 to 39	7·1%	8·0%	1·2%
40 to 49	7·5%	9·0%	4·1%
50 to 59	22·6%	15·0%	5·5%
60 to 69	32·1%	17·0%	12·5%
70 and over	43·3%	32·0%	21·3%

and partly by limiting the scope of successful surgical treatment. Certain factors such as sex, history of previous hæmatemesis, and length of history, do not influence the mortality (Lewin and Truelove, 1950).

Thus the important factors contributing to a high mortality are a chronic ulcer (particularly gastric), recurrent bleeding after admission to hospital, increasing age, and the presence of associated complications. Before the question of surgical treatment is considered, the role of bleeding in acute gastric lesions will be further analysed.

Acute Gastric Lesions Causing Bleeding.

It has been found that a third of the patients admitted to hospital for hæmatemesis and melæna fail to show any radiological evidence of peptic ulcer and other disease. Over half of this group have undergone a gastroscopic examination, usually between the third and the tenth days after admission to hospital, and acute gastric ulcers have been demonstrated in 40% of those so examined. In some cases there is possibly diffuse gastrostaxis and no definite ulcer, and in two cases a small acute ulcer has been seen at the site of a previously observed intramucosal hæmorrhage.

These acute ulcers seen gastroscopically did not give rise to severe epigastric pain, and the patients may have had either no discomfort or a history of epigastric pain for a variable period before admission to hospital with hæmorrhage. These ulcers have usually healed within two to four weeks, but have not shown any evidence of their presence radiologically when the patients have been radiologically examined while convalescent. The bleeding may be severe and recurrent for these acute lesions. Admittedly the comparison is not a strict one, as more patients with chronic ulcers have been operated on, but the comparable severity of bleeding between the "acute" groups with no X-ray evidence and the "chronic" gastric ulcer groups with X-ray evidence is shown in Table IX.

Approximately one-quarter of the patients in the acute lesion groups have had recurrent bleeding after their admission to hospital. However, the mortality rate has been low except in those aged over sixty-five years.

These patients with acute gastric ulcers diagnosed gastroscopically have been carefully followed up, and their cases are being reported elsewhere (Avery Jones and W. E. King). The men tend to return later with characteristic gastric ulcers. The women appear to have a more benign prognosis, and although further episodes of bleeding may occur there is little tendency for gastric ulcers to develop. With the previous considerations in mind the question of surgical treatment will be discussed, and I should like first to pay a tribute to the pioneer work of Sir Gordon

TABLE IX.
Lowest Hæmoglobin Reading.

Hæmoglobin Value.	Chronic Gastric Ulcer. (Percentage of Subjects.)	Gastroscopically Verified Acute Gastric Ulcer. (Percentage of Subjects.)
20%	9·6	6·0
30%	16·2	16·9
40%	19·8	20·5
50%	19·8	27·7
60%	13·5	15·7
70%	7·7	6·0
80%	3·6	2·4
90%	1·8	1·2
100%	1·8	—
Not known ..	6·3	3·6
Total	100·0	100·0

Gordon-Taylor, who demonstrated that massive hæmorrhage from chronic ulcer could be treated successfully by emergency operation if it was carried out promptly. At that time surgery had fallen into disrepute, as it had been demanded as a last resort for patients who were grossly anæmic and anoxæmic and often dehydrated. However, the introduction of liberal feeding by Meulengracht (1935) and of drip transfusion by Marriott and Kekwick

TABLE X.
Acute Gastric Lesions, 1941 to 1951.

Age (Years).	Male Subjects.	Female Subjects.
25	4	8
25 and over	19	22
35 and over	42 (1 died)	25
45 and over	39	52
55 and over	44	39
65 and over	38 (5 died)	30 (3 died)
75 and over	7	9
Total	193 (6 died)	185 (3 died)

(1935) greatly improved the medical care of these patients. To try to assess the contribution made by liberal feeding and drip transfusions, I made a study of 400 consecutive patients admitted to hospital with gastro-duodenal hæmorrhage and treated medically with a minimum of surgical intervention, and was convinced that there was scope for immediate operation in a proportion of cases. Our experiences of the indications for surgical treatment may be summarized as follows.

1. The surgical risk is greatly increased by the presence of serious medical complications, and I would particularly point out gross obesity, a severe degree of emphysema with chronic bronchitis and copious sputum, recent coronary infarction, calcareous aortic stenosis, pyelonephritis particularly associated with nervous disease, and heart failure; in such cases the mortality rate is very high. If one is satisfied that the medical and surgical mortality will be of the same order, surgical intervention is not justified.

2. The best surgical contribution to the reduction of mortality comes between the ages of forty and seventy years. The mortality from recurrent bleeding in patients aged under forty years is much less than in the older age range, but nevertheless the occasional young patient may die, and operation must not be refused after the second severe recurrent hæmorrhage in hospital. Similarly the upper age range must be elastic. A presenile individual, aged sixty-five years, leading a vegetative existence, presents a more serious hazard than an alert, active man or woman aged eighty years.

3. Recurrent hæmorrhages after admission to hospital in a patient known to have a chronic gastric or duodenal ulcer are an indication for surgical treatment, preferably partial gastrectomy. It is not easy to define "chronicity". Ulcer-type pain, particularly if severe, for a few weeks is an adequate indication. Less pain, but with a history of

FIGURE II.
Gastroscopic drawing showing two acute gastric ulcers.

a previous radiological diagnosis of gastric or duodenal ulcer even after a long remission, may be accepted.

4. Patients with known chronic ulcer, from whose history a partial gastrectomy is clearly indicated, or who may already be waiting admission to hospital for operation, should be treated surgically. It has been the exception rather than the rule for immediate operation to be performed on patients with previous known ulcers, unless further bleeding has occurred in hospital. Nevertheless, on occasions immediate operation may reasonably be recommended.

5. Most patients with little or no ulcer dyspepsia, who have recurrent bleeding after admission to hospital, remain a debatable group. In such cases the bleeding is due to a superficial acute ulcer or to a widespread gastrostaxis. But occasionally painless chronic ulcers may exist, or bleeding may be due to a simple tumour. Our policy has been to perform a gastroscopic examination after the first recurrence of bleeding and to consider laparotomy after the second or third recurrence, and this may be particularly necessary if the blood supply is limited; but with the low mortality rate in patients aged under sixty years a medical bias is justified. It may be necessary to open the stomach to locate the ulcer. In women the follow-up prognosis does not justify a partial gastrectomy for a small acute ulcer, and for preference a local submucosal removal of the ulcer should be undertaken. In men the follow-up prognosis is poor, and partial gastrec-

tomy is a reasonable procedure. Our policy is against performing a blind gastrectomy if no ulcer is found; but it may occasionally be justifiable in the treatment of men with very severe recurrent bleeding.

6. Patients with simultaneous or consecutive hæmorrhage and acute perforation of a peptic ulcer should be considered for emergency partial gastrectomy.

7. For patients with evidence of pyloric stenosis, operation should be considered before further bleeding occurs, but a suitable interval should be allowed for restoring the body fluids and electrolytes.

TABLE XI.
Mortality by Age and Period.

Age. (Years.)	1941 to 1945.		1949 to 1951.	
	Cases.	Deaths.	Cases.	Deaths.
All Patients with Bleeding Treated Surgically.				
Under 40	85	1 (1·2%)	76	1 (1·3%)
40 to 64 ..	275	13 (4·7%)	263	10 (3·6%)
65 and over ..	85	14 (16·5%)	117	27 (23·1%)
Patients with Recurrent Bleeding from Chronic Gastric Ulcer Treated Surgically.				
Under 40 ..	—	—	3	—
40 to 64 ..	20	9 (45·0%)	24	3 (12·5%)
65 and over ..	5	3 (60·0%)	14	6 (43·0%)
Patients with Recurrent Bleeding from Duodenal Ulcer Treated Surgically.				
Under 40 ..	3	1 (33·0%)	5	1 (20·0%)
40 to 64 ..	13	2 (15·0%)	23	3 (13·0%)
65 and over ..	7	3 (43·0%)	11	4 (31·0%)

8. Patients who have previously undergone gastro-enterostomy or partial gastrectomy present a special problem. Their reputation for offering less hazard than patients with a known gastric or duodenal ulcer is probably due to the fact that in many cases the bleeding is due to an acute lesion of the stomach or jejunum causing jejunitis or acute ulceration, rather than to penetrating ulcers. They carry a very low mortality rate from medical treatment. They usually have had little or no recurrent dyspepsia. When there has been considerable recent pain, or when a definite stomach or duodenal crater has been demonstrated, they should be treated as for any other chronic ulcer.

This surgical policy has been in operation at the Central Middlesex Hospital since 1945. A comparison of the mortality in successive years has not actually shown a fall in mortality, but these figures are fallacious, as there has been a much higher proportion of elderly patients in the later years. A critical analysis of the mortality figures in different groups has demonstrated that the best results from this surgical policy have been obtained among patients with recurrent bleeding from chronic gastric ulcer. There has been some fall in the corresponding figure for duodenal ulcer.

The mortality in the over sixty-five years age group has actually increased, and it is likely that particular discretion is needed in the selection of patients for surgical treatment in the older age groups if the overall mortality is to be reduced among them (Table XI).

Medical Management.

The best results can be obtained only by well-organized team work among physicians, surgeons and pathologists. There should be a measure of agreement between the senior members of the staff on the principles of treatment, and these should be set out in the schedule available as a general guide for junior medical and nursing staff. The duty surgeon should be notified of any patient who comes within a definite group in which surgical treatment may be considered advisable if bleeding occurs. The same surgeon may wish to examine all ulcer patients admitted, but this is seldom practicable unless the hospital is exceptionally well staffed.

When a diagnosis of hæmatemesis or melæna is established, early reassurance of the patient is essential. If necessary, this may be augmented by barbiturates or by morphine. It is important to realize that morphine may be lethal to patients with severe emphysema and its routine use is not recommended. A medical history must be taken and an examination made as soon as possible, and the severity of the illness must not be used as an excuse for delaying this step, which may be essential in deciding subsequent treatment. The relatives should also be interviewed, and not infrequently they may be able to add useful points to the history; the relatives also need reassurance, and the opportunity to enrol them for the blood bank must not be missed.

FIGURE III.
Drawing of partial gastrectomy specimen showing double chronic gastric ulcers, one having perforated and the other having bled.

Five millilitres of blood should be obtained for initial hæmoglobin value estimation, blood urea estimation and blood grouping tests, including rhesus testing, and for serum for direct testing if transfusion is necessary.

The members of the nursing staff are instructed to record the pulse rate hourly, and the blood pressure should be recorded if there is increased tachycardia. The patient is allowed a semi-solid *purée* diet, with two-hourly feeds of seven ounces of milk food if desired; the majority of patients will prefer taking food if it is offered to them. Thirst must be entirely prevented by allowing as much one-third normal saline, flavoured with fruit juices, as desired. The next decision relates to blood transfusion.

It is difficult to state precise indications for blood transfusion. It must be remembered that bleeding, if recurrent, tends to take place at intervals of twelve to thirty-six hours, and that transfusion is needed not so much for the first hæmorrhage, which is seldom fatal, as to enable the patient to withstand further bleeding should this occur. The giving of blood at the rate of one pint in four hours by drip transfusion will restore a reserve, but more rapid transfusion may be given if there is evidence of continued

bleeding. It is essential to prevent the development of anoxæmia, for this is particularly liable to lead to restlessness and lack of cooperation at all times and to coma and irreversible shock in the elderly; again, bleeding in an already anæmic subject invites the small but appalling risk of permanent blindness. If bleeding has clearly been severe, if the patient is still shocked, if there is persistent tachycardia, and if the hæmoglobin value has already fallen to 50%, transfusion should be begun. The risk of raising the venous pressure and precipitating acute heart failure must be kept in mind when a transfusion is given to anæmic patients, and the jugular venous pressure must be under observation during the transfusion. It is worth while having an indwelling small stomach tube passed through the nose, a milk food may be given by the continuous drip method, and samples of gastric juice may be withdrawn hourly. Patients who become desperately ill should be given oxygen, preferably with an oxygen tent. In severely ill patients the stomach may become very distended, and aspiration with an œsophageal tube and a stomach pump may improve the general condition. If gastroscopy is required to locate a bleeding point, this investigation can be undertaken in the ward without the patient's being moved from his bed. Emergency radiography in our experience is seldom helpful, and barium in the bowel may add to post-operative difficulties. A barium meal examination is best left until the patient can stand without the risk of fainting—that is, in the second or third week after admission to hospital. In cases of suspected *cirrhosis hepatis* a careful clinical examination must be made for spider nævi and for hepatosplenomegaly, and in my experience the diagnosis is seldom in doubt in suspected cases; bromsulphthalein may give valuable assistance. The stools should be examined for occult blood in the second or third week; should the result remain unaccountably positive, X-ray examination with a barium enema is advisable to exclude a coexisting neoplasm. The combination of bleeding duodenal ulcer with carcinoma of the colon is an occasional cause of admission to hospital for melæna. During the days after admission it is desirable to give an antacid—for example, "Aludrox", two teaspoonfuls six times a day after feeds—and phenobarbitone, 0·5 to 1·0 grain two or three times a day, to maintain mental relaxation. The bowels are usually constipated, but no action should be taken other than reassuring the patient and giving a simple enema after four days. Iron should be given by mouth when melæna has ceased.

SUMMARY.

1. An analysis is presented of the ulcer admissions to Central Middlesex Hospital for acute perforation and bleeding.

2. The evidence suggests that immediate partial gastrectomy has a proper place in the management of selected patients with acute perforation.

3. The indications for surgical intervention and details of medical management of bleeding peptic ulcer are discussed.

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THE MANAGEMENT OF THE COMPLICATIONS OF PEPTIC ULCER.[1]

By SIR GORDON GORDON-TAYLOR, K.B.E., C.B.,

Consulting Surgeon, Middlesex Hospital, London, and the Royal Navy.

IT is an honour which I greatly prize to have been invited to participate in a debate in this city, which I have long loved, and before an audience for many of whom I entertain a great affection, and on a subject which has been of very considerable interest to me for exactly forty years (it was in 1912 that I first operated on a patient with ulcer hæmorrhage at the request of the late Dr. Birkdale-Morison at the Royal Northern Hospital); and the honour and pleasure are made all the greater by my association in today's discussion with my good friend Dr. Francis Avery Jones, whose high reputation in Britain as our foremost gastro-enterological physician must have already found the most ample confirmation in this land since his arrival in your midst. This assembly at any rate affords me an opportunity of expressing *coram publico* my gratitude to him for abundant help and innumerable kindnesses over many years, not only in the treatment of naval personnel—for we have both a consultant status to the Royal Navy—but also in the management of civilian gastro-enterological problems and in connexion with my own Lettsomian Lectures delivered a few years ago, when he magnanimously put the whole of his even then vast material at my disposal and invited my criticism of his methods and his results.

I might in truth have entitled my remarks today as "Looking Backwards" or "In Retrospect", since all that I have worked for and hoped for since 1919 in connexion with the treatment of ulcer hæmorrhage has eventuated, and the surgeon is no longer excluded from consultation until that dire stage in the dismal downward progress of the case of ulcer hæmorrhage has been reached when the patient is worn out with hope deferred, his tissues are poisoned by morphine, his biochemistry is altered by the starvation enjoined in those days of the "dark backward", and his cough reflex is abolished by hypnosis, so that the stage is all set for the terminal bronchopneumonia which in former days so frequently closed the scene.

If realism is the first condition of wisdom, it is passing strange how many of the wise physicians of twenty and thirty years ago could deceive themselves as to the mortality of ulcer hæmorrhage; a visit to the post-mortem room might surely have suggested an earlier appeal to surgery. I doubt if there are any here today who were present at a memorable meeting of the Medical Society of London when this very subject of today's discussion ranged against me a phalanx of physicians like Sir Arthur Hurst, Professor John

[1] Read at a combined meeting of the Section of Medicine, the Section of Surgery and the Section of Radiology and Radiotherapy, Australasian Medical Congress (British Medical Association), Eighth Session, Melbourne, August, 1952.

Ryle, Sir Henry Tidy, Sir Edmund Spriggs and others who fiercely assailed me for suggesting such a heterodox method of treatment as surgery, and it was confidently asseverated that these patients with ulcer hæmorrhage never died. Sir Arthur Hurst, I know, was ultimately persuaded, but Ryle remained to the end, I believe, a recalcitrant disbeliever in the lethal termination of the condition. I would not waste your time for a moment by reminding you of fantastic statements made by physicians that blood transfusion as employed not so many years ago probably cost more lives than it saved, or that fatal cases of ulcer hæmorrhage never occurred in private practice, or that blood transfusion was scarcely ever required or enjoined for this seemingly favoured class of sufferer. Nevertheless, during the years 1924 to 1933 many of my own fatal cases (in which, alas, non-operative measures were employed) belonged to that very group in which the future was alleged to be so rosy.

The pioneer is rarely the exploiter, and the man who destroys an old edifice and marks out the lines of a new one does not often live to see the walls arise. In this respect I have indeed been fortunate; my plea for surgery in the appropriate case, so often made and reiterated, has survived the frosts of the most hostile criticism of a previous generation of physicians and the internecine battles with those who fiercely resented the impertinent promulgation of a surgical doctrine for a malady regarded from time immemorial by the physicians as peculiarly their own, and I have been spared to see a closer medico-chirurgical *rapprochement* and a wise and loyal cooperation in dealing with this problem of hæmorrhage from gastro-duodenal ulceration.

It may be impertinent for an old surgical gladiator like myself to emerge once again in "the sere and yellow" of my life, but I am flattered that you do not regard me as one who has been given the stave of gladiatorial retirement—in the words of Horace, "*spectatum satis et donatum iam rude*" or as one deserving the "beach" or "the bowler hat".

It is true that in my tentative and less certain efforts during the decade that immediately followed the first Armageddon changes in outlook from time to time controlled my scalpel, but no surgeon worthy of the name remains intransigently and adamantly bound to some particular line of therapeutic conduct which he may have cherished or championed heretofore, nor should his attitude toward any problem be crystallized beyond the power of reasoned persuasion. In the treatment of the hæmorrhage of peptic ulceration my own later experience and the far greater experience of others interested in the problem have modified some views formerly entertained by me, have clarified difficulties, have dispelled the mists of doubt and uncertainty and have matured judgement.

Horace in his "*Ars Poetica*" advises the would-be poet to put his manuscript aside for nine years before publication: I allowed twice that period of time to elapse before my first appeal for medico-chirurgical cooperation, which therefore was not idly or thoughtlessly advanced. I may lack the prophetic vision of a Cassandra, but long years ago I ventured to write on two occasions as a result of my experience in the following terms:

> It may be long before the physician and surgeon see absolutely eye to eye as to the necessity and as to the appropriate moment for operative interference, but I have no doubt whatever that ultimately the internist will become more alive to the menace of disaster which threatens the patient and will become more surgically minded towards these cases than he may be at the moment. Surgery will then inspire in him the same hope of arresting the hæmorrhage of peptic ulcer as of saving the life of a ruptured ectopic gestation.

And again I wrote:

> I am still hopeful that the time may come when medical science will of a certainty cure every ulcer of the stomach or duodenum without recourse to surgery, but until that golden era ulcer hæmorrhage, perhaps severe ulcer hæmorrhage, will continue to occur and recur, gravely menacing life and perchance ending disastrously. *The problem of the bleeding peptic ulcer is certainly no longer one of surgery versus medicine,* although without a doubt there are cases which operation alone may save. That a predominant percentage of ulcer hæmorrhages cease spontaneously or by reason of medical treatment, to which blood transfusion may have been added, has never been denied by myself; it is also freely admitted that the success which has followed early operation might well have attended an expectant line of treatment; but it is no less true that late surgical intervention consequent upon unsuccessful medical therapy is associated with mortality; therein lies the dilemma in treatment.
>
> I am merely pleading once again that the surgeon be not excluded from the consultation until that last desperate stage of ulcer hæmorrhage, the anxieties of which neither physician nor experienced operator is anxious to shoulder. *Every case is to be regarded as a special problem deserving of the most careful attention.* Supreme judgement on the part of the surgeon will be of more value to his patient than the acme of technical skill; indiscriminate operation cannot be too strongly condemned.

Although the golden era of the cure of every ulcer by non-operative means has not yet dawned, the physician and surgeon are today a loyal team.

Suggested Outline of Treatment and Indications for Surgery in Cases of Hæmatemesis.

1. In every case of hæmatemesis or melæna, whatever the cause, immediate steps should be taken to organize donors from amongst the relatives and friends or obtain access to a blood bank, so that a sufficiency of blood of the same or a suitable group can be relied upon, should necessity arise. It is impossible to foretell what may happen in a case of ulcer bleeding.

2. In every case in which bleeding is more than slight—that is, when the rate is 100 per minute or more and the hæmoglobin value is, say, 60%, a drip transfusion should be commenced. A pulse rate which remains over 100 per minute indicates a great loss of blood and a risk for any prospective surgical treatment; a patient with a hæmoglobin value of only 50% is in a relatively poor state to stand a resection, should such be deemed necessary at the time of operation. The need for drip transfusion for such patients is manifest.

3. It is desirable that every patient who has had a hæmatemesis should be transferred to a fully organized and equipped institution, and if there is a possibility that the hæmorrhage originates from a gastric or duodenal ulcer the necessity for immediate transference is the more imperative. The exsanguinated patient can be removed with the drip transfusion running when this seems indicated. The objection was raised by physicians in the past that patients with grave gastro-intestinal bleeding should never be moved, that the vast majority of patients with hæmatemesis seen in hospital should never have been sent for admission, and that such patients had been transferred thither through mistaken judgement on the part of their medical adviser, although in certain instances bad home conditions may have indicated the step. The employment of drip transfusion and the results thereof during transportation impose the necessity of reconsidering this enjoinment; patients have been transferred to me in London by ambulance, and even in a guard's van, with the drip transfusion runing, for distances up to 80 miles, and despite the journey have suffered no set-back on the road. One patient, bleeding from an ulcer and having a hæmoglobin value of only 30% on departure from a town 60 miles away from London, arrived at hospital with a better blood picture as a result of the continuous transfusion ("travelling transfusion") than when he started on his long journey. The objections raised on the score of the grave danger of transport may thus be contested in the light of certain experiences. If transportation is decided upon, it should be undertaken early; any further complication, such as a subsequent perforation *et cetera*, would preclude removal, especially over a distance of any magnitude.

4. There must be early consultation between physician and surgeon, for which I have pleaded for so many years, fortunately now with success.

Indications for Operation.

1. Recurrent hæmorrhage in a patient aged over fifty years, known clinically and radiologically to have a chronic

gastric or duodenal ulcer which has bled before, should indicate operation. On the other hand, the diagnosis of a chronic ulcer may have to depend on the history of characteristic pain, severe in character and lasting for some weeks, without X-ray evidence; or it may rest upon a less severe history but on positive radiological evidence, even if there has been a long period of remission of symptoms and a long interval since the X-ray examination.

2. If such a patient, aged over fifty years, under expert treatment in an institution, has further bleeding or if bleeding continues on the second day, urgent surgical treatment is indicated, since the fatal results of bleeding and the increasing risk of operation for bleeding on the third day are to be remembered (Finsterer, Tixier and Clavel, Gordon-Taylor).

3. Patients who have hæmatemesis or melæna with little or no ulcer dyspepsia, but who have recurrent or persistent bleeding after admission to hospital are unlikely to die from a single hæmorrhage, but operation is justified after a second or third bleeding.

4. Patients with simultaneous or consecutive hæmorrhage and acute perforation should be considered for an emergency partial gastrectomy. Hæmorrhage may occur at any time in the first ten days or even later in a small proportion of those operated on for perforation. Such patients do badly,[1] and an emergency partial gastrectomy is to be recommended; in some either a double chronic ulcer or a giant single ulcer or a generalized gastrostaxis may be found.

5. For patients with evidence of pyloric stenosis or mid-gastric narrowing (Gordon-Taylor) operation should be considered before further bleeding occurs, but if possible a suitable interval should be allowed for restoring the body fluids and electrolytes.

6. Patients with a previous gastro-enterostomy or gastrectomy constitute a special problem. Their reputation for presenting less hazard than those with a known gastric or duodenal ulcer is probably because in many cases there is an acute lesion of the stomach or of the jejunum (jejunitis or acute ulceration) rather than a penetrating ulcer. Such cases carry a very low mortality on medical treatment. They have usually had little or no recurrent dyspepsia. When, however, there has been considerable pain or when a definite ulcer crater has been already demonstrated in the stomach, duodenum or jejunum, they should be treated as for any other chronic ulcer.

7. Every case has to be considered on its own merits; there can be no universal rule. Intercurrent illness, the previous habits and physical conformation of the patient, the personal courage of the patient as well as of the operator, the environment, the technical skill and experience of the surgeon, may all be factors which not only influence physician and surgeon in arriving at a decision regarding the desirability of surgery, but may also dictate the appropriate technique, when the abdomen has been opened.

8. The surgical risk is of course greatly increased by complications, such as a severe degree of emphysema, chronic bronchitis with copious sputum, recent coronary infarction, calcareous aortic stenosis, cerebral vascular disease, pyelonephritis, especially associated with nervous disease, obesity, bronchopneumonia, heart failure, cachexia, which may sometimes be associated with very large ulcers in undernourished people, drug addiction (Avery Jones).

9. The time factor is of importance as regards both the precise interval between the onset of the hæmorrhage and the hour of surgical interference, as well as the duration of the operative procedure necessary to arrest the bleeding. It is well known that the "hæmorrhage period" in cases of bleeding chronic peptic ulcer usually lasts for several days, and that it is the rapid recurrence or persistence of bleeding which occasions anxiety and which may prove fatal. Long ago, Tixier and Clavel, analysing the cases of ulcer in which death occurred from hæmorrhage, found that in 40% the fatal termination was reached within three days and that in 25% death occurred in the third day itself. For these authors, as for Finsterer, the third day was the critical day. There can be no room for doubt that early operation for ulcer hæmorrhage, as for perforation, still holds out the highest hope for those patients for whom surgery is indicated, and Finsterer's "first forty-eight hours" is still a signpost to guide the way. Personal experience, however, in this late period of my own life, and the vastly greater recent experience of other surgeons, convince me now of the possibility of recovery from operations performed much later than the first forty-eight hours, which once seemed to be the dividing line between success and failure. The difficult dilemma of earlier years of an urgent decision as to the need for surgery has therefore been somewhat simplified. Tanner wisely concludes as follows: If one operates late, the mortality will be high; if one operates early, it will be lower; but either procedure will be correct if it produces the lowest over-all mortality.

10. The dexterity and speed of the surgeon must influence not only the decision to advise surgical treatment, but also the result. R. L. Galloway, formerly of Chase Farm Hospital, London, an experienced and skilful operator, believed that urgent surgical treatment for ulcer hæmorrhage should be carried out only by "someone who is nimble with his hands and can carry out the operation at the maximum possible speed". Ivor Lewis, late of the North Middlesex Hospital and now of North Wales, thought that no surgeon should operate in a case of bleeding ulcer during the "hæmorrhage phase", in which the mortality for cold gastric surgery is more than 5%. Norman Tanner is no surgical tortoise, nor are they operative sluggards or sloths at the Central Middlesex Hospital, to whom Avery Jones delegates his surgery.

I have always employed regional and splanchnic anæsthesia for these cases of hæmorrhage, following the teaching of Finsterer. Norman Tanner follows the same rule.

Percentage of Patients Submitted to Surgery.

The percentage of patients with ulcer bleeding submitted to surgery *à chaud* varies enormously in different clinics even today. In the most recent statistics (Ogilvie, Cardoe and Bentley) from the Royal Victoria Infirmary, Newcastle-on-Tyne, 10% of the patients admitted for massive ulcer bleeding came to operation. At the Royal North Shore Hospital, Sydney (J. C. Fitzherbert and R. G. Epps), 6·8% of such patients underwent surgical treatment. Norman Tanner's policy has varied over the years, and the percentage of patients with ulcer hæmorrhage operated on by him has ranged from 5% to 60%. In my own smaller though protracted experience my percentage of patients with hæmorrhage submitted to surgery has been about one in six (16·6%).

Fatality of Operation for Ulcer Hæmorrhage.

The variables are so numerous in comparing the operative mortality rates of ulcer hæmorrhage in the hands of different surgeons that any comparison would be invidious and worthless, but the following are random examples of fatality percentages: Bohn (of Reading), 6%; Parsons and Aldrich, 14%; Gordon-Taylor, early cases 3%, late cases 30%; Hans Finsterer, early cases 4%, late cases 22·8%; Norman Tanner performed 110 operations with the remarkably small mortality rate of 7%—especially remarkable when it is to be remembered that his series comprised the most aged group of patients yet recorded, and the average age of the patients who died was no less than sixty-six years.

The Nature of the Surgery Employed.

Although those surgeons who have had most experience of operation during the phase of acute hæmorrhage from chronic peptic ulcer today exhibit a proclivity towards the performance of partial gastrectomy with or without duodenectomy according to the position of the ulcer, in my own hands the treatment of ulcer hæmorrhage has never been synonymous with gastrectomy. Perhaps surgeons are more skilful and dexterous today.

[1] Norman Tanner, whose skill and experience are well known, has produced encouraging figures for "secondary gastrectomy" for what is often a calamitous complication of a successful closure.

There is no one technique appropriate to every case for which operation has been judged necessary. The object of surgery is to arrest hæmorrhage, and I have at times availed myself of such varied surgical measures as the following: gastrotomy or duodenotomy, with ligature of the bleeding vessel or vessels from the mucous membrane aspect; pyloric occlusion and gastro-jejunostomy; cauterization or diathermy of gastric ulcer and gastro-jejunostomy; sleeve resection; gastrectomy, local or partial; gastro-duodenal resection; Finsterer's "resection for exclusion"; excision of duodenal ulcer with some form of plastic operation on the pylorus; and even jejunostomy on rare occasions. The ligation of the gastro-duodenal artery in continuity in cases of bleeding duodenal ulcer has been found a useful adjunct to such indirect operative attack as pyloric occlusion and gastro-jejunostomy, but in the case of callous duodenal ulcer with much surrounding œdema or infiltration the exposure and control of the vessel by ligature will be difficult.

In the case of bleeding anastomotic ulcer I have practised complex and complicated resections, and I have also dealt with a bleeding gastro-jejunal or jejunal ulcer by an approach through the anterior stomach wall. Fortunately the bleeding of anastomotic ulcer is rarely fatal, and one can more safely postpone surgical interference. Despite the greater uncertainty of indirect operation as a means of securing hæmostasis in bleeding duodenal ulcer, some of my greatest successes have followed a mere gastro-jejunostomy with pyloric occlusion. A bleeding duodenal ulcer treated by exclusion may occasionally bleed once after this type of operation, but in my experience it has never bled recurrently.

As an effective method of hæmostasis I believe jejunostomy to be unreliable and to be avoided, except in the desperate case; but jejunostomy certainly saved the lives of two elderly women with high gastric ulcers, and one of them today, nearly ninety years of age, survives twenty years after the incident.

Hæmorrhage and Perforation.

In previous communications I have written at some length on my own experience of the association of perforation and hæmorrhage occurring synchronously or successively, and expressed my own view that it might sometimes be in the best interests of the patient that these twin complications of peptic ulcer should happen together. Nothing has emerged from the untoward experiences of other surgeons to dissipate the dismal prognosis attaching to those patients whose ulcers bleed and thereafter perforate or perforate and subsequently bleed.

At Middlesex Hospital a patient of H. L. McMullen within a few weeks suffered two hæmorrhages and two perforations of a duodenal ulcer. He was admitted to hospital as an in-patient for severe hæmatemesis, and his ulcer perforated ten days later; the perforation was successfully sutured. A fortnight later another hæmorrhage occurred, and during the period of recovery from this a second perforation took place, to which the patient succumbed.

A history such as this lends support to the practice of primary gastro-duodenal resection in the treatment of perforated peptic ulcer long advocated by Professor S. S. Yudin, of Moscow, in his well-organized emergency hospital (Sklifassovsky Institute), with its excellent first-aid and wonderful transport facilities, and also by many Continental surgeons, and nowadays (in selected cases) by British surgeons.

Summary.

1. Results confirm the wisdom of the segregation of cases of severe ulcer hæmorrhage under physicians who display a special interest in the problem.

2. My own contribution to the treatment of ulcer hæmorrhage has been mainly provocative, for I feel sure that by my advocacy of surgery in selected cases I did something to reawaken the interest and to disturb the complacent attitude of an older generation of physicians towards a subject hitherto regarded by them as peculiarly their own. Since 1935, which saw the introduction of the Marriott-Kekwick "drip" method of blood transfusion and the institution of the Meulengracht method of early feeding, the results of the non-operative treatment have vastly improved and are no longer a reproach to medicine.

3. Today I am no longer as the voice of one crying in the wilderness of intransigent medical conservativism; a change of heart towards the physicians' surgical colleagues is becoming increasingly manifest, and the aid of surgery is often sought, and today is happily sought early.

4. In the evolution of this fraternal cooperation the post-graduate hospitals of London, especially those of the old Middlesex County Council and the London County Council, have led the way. In these the team spirit between physician and surgeon first came to fruition. I would be lacking in my presentation of the subject if I did not mention the names of friends, such as Avery Jones and his surgical confrères; Norman Tanner, who combines the role of physician and surgeon, and his younger colleagues like Desmond and Craig (now of Lowestoft); Galloway and Birch; and Ivor Lewis, of Chase Farm Hospital (now of North Wales).

TABLE I.

Summary of 86 Operations Performed during Hæmorrhagic Period for Bleeding Chronic Peptic Ulcer (Personal Series).

Period.	Operations.	Recoveries.	Deaths.	Percentage Mortality.
1919 to 1924 ..	22	20	2	9
1924 to 1926 ..	10	6	4	40
1926 to 1933 ..	10	8	2	20
1933 to 1939 ..	Early operation 18	17	1	5·5
	Late operation 11	7[1]	4	36
	29[2]	24	5	17
		24	3	11
1939 to 1952 ..	Early operation 13	13	0	0
	Late operation 2	2	0	0
1933 to 1952 ..	44	39	5	11
	42[3]	39	3[3]	7[3]

[1] Two of the four fatalities in the late operations were hopeless cases in the 1933–1939 series.

[2] Total 29, but amended total 27 (two hopeless cases excluded).

[3] Two hopeless cases excluded.

5. It is refreshing at the beginning of this decade no longer to read statistics which deal with the results of surgery in the treatment of ulcer hæmorrhage, or with the recovery rate of those for whom non-operative measures have been employed, but to have attention directed to the "over-all mortality".

6. Therapeutic opinion appears to vary as to the value of computations of blood volume in estimating the severity of blood loss not only in the early phase of ulcer hæmorrhage, but more especially in recurrent bleeding. Considerable store was set upon this by Izod Bennett and his colleagues at my own hospital in the treatment of patients with severe ulcer hæmorrhage; an estimate of the calibre of the artery bleeding in the base of the ulcer was sometimes tentatively and correctly adumbrated. Doubtless, gastroscopy furnishes a more accurate picture and substitutes certainty for guesswork.

7. The influence of age upon mortality is indeed striking, and any comparison of statistics of individuals, of institutions or of methods of therapy which do not take this into account is worthless (Allen and Benedict; Avery Jones *et alii*). In Tanner's series the over-all mortality rate was 1·5% for patients with hæmorrhage aged under fifty years and 13·8% for those over that age. Avery Jones found it to be 2% for those aged under forty-five years and 6% between the ages of forty-five and sixty years, increasing to 12% for those in the sixties and to 21% for septuagenarians and the Methusalehs of the population. Why does age engender ingravescent mortality? For three reasons: (*a*) the increasing rate of gastric and duodenal

ulcer with advancing years, associated with the known higher mortality of the former; (*b*) the increasing incidence of arteriosclerosis in older patients, with its local and general effects; (*c*) the growing incidence of concomitant disease—cardiac, renal, cerebral *et cetera*—natural to advancing years.

The Treatment of Perforated Peptic Ulcer.

It is a wonderful experience to have witnessed the decline in the mortality of perforation of a peptic ulcer from 50% in the first decade of the century to the infinitesimal figure of today. The "time-lag" between the catastrophe and operation has played the most important role in this decline, but anæsthesia and antibiotics have been of paramount value. From the Central Middlesex Hospital figures up to 1948 the mortality was less than 4%, and between 1948 and the end of 1950 only three died out of 160 patients treated by suture or gastrectomy—less than 2%. Ian Aird had four deaths in 73 cases (5% mortality); Brian Truscott and J. F. R. Withycombe had a similar mortality from suture at Addenbrooke's Hospital, Cambridge. Andrew Lowdon, from the Royal Infirmary, Edinburgh, dealt with 65 patients, of whom 51 were submitted to primary partial gastrectomy, with a mortality of 6%, none of the gastrectomy patients succumbing. Paterson Brown, of Edinburgh, had a series of 50 cases of perforated ulcer without a death. Increasing confidence in surgery has led to an extension of the operative procedure in cases of perforated ulcer. Following in the wake of Continental operators, surgeons in Britain have been performing primary partial gastrectomy in selected cases of perforated gastric and duodenal ulcer (Andrew Lowdon, the surgeons of Central Middlesex Hospital *et alii*).

I have little or no experience myself of primary gastrectomy for perforation, save when there is concomitant hæmorrhage; but Lowdon is of the opinion that immediate gastrectomy should not be performed on a patient who has only a short history of dyspepsia and is found at operation to have a perforated duodenal ulcer of an acute or subacute type.

In his opinion, gastrectomy should be performed under the following conditions: (i) in cases of chronic or recurrent ulcer, which perforates; (ii) in cases in which there is a short history of dyspepsia, if (*a*) there has been a previous perforation, (*b*) hæmorrhage accompanies the perforation, (*c*) the perforation is gastric, (*d*) the findings at operation indicate that the duodenal ulcer is of a definitely chronic type. It need not be stressed in this assembly that the decision to perform gastrectomy will also be influenced by such factors as the "time-lag", the general condition of the patient, *et cetera*.

In view of the wonderful results obtained today in the surgical treatment of perforation, he must be a brave man who would champion non-operative measures as an alternative to surgery. The conservative method has, however, a definite place in the following circumstances: (*a*) when the diagnosis is in doubt between a perforation and some condition in which operation would be contraindicated—for example, coronary thrombosis, diaphragmatic pleurisy, *et cetera*; (*b*) when the condition of the patient, whether as a result of the perforation or other causes, makes operation inadvisable; (*c*) when signs are indefinite and a large perforation with much soiling of the peritoneal cavity is improbable; (*d*) when good surgical facilities are not immediately available, as in ships at sea *et cetera* (Truscott and Withycombe).

Pyloric Stenosis and Mid-Gastric Narrowing.

Gastrectomy is *à la mode* today, but I would most earnestly warn against the performance of gastrectomy in women, unless there is a suspicion of malignancy or unless a gastrectomy appears to be the safest procedure in a particular case—a rare contingency. Anastomotic ulcer is also rare in women and therefore constitutes a very infrequent indication for a possible gastrectomy. The degree of anæmia, which in my experience is always microcytic, may be extreme in women who have undergone gastrectomy, and is never slight, and some of the most distressing results of my surgical life have concerned these unfortunates. Happily they are few in number, for I learnt my lesson a quarter of a century ago. In these days the very safety of surgery tempts the younger operator to perform gastrectomy lightheartedly; it should be a criminal offence to perform gastrectomy on a woman, except under special circumstances, although it is admitted that patients may react in diverse manners in different territorial environmental conditions.

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THE MANAGEMENT OF PEPTIC ULCERATION.[1]

By JOHN O'SULLIVAN,
Melbourne.

I REGARD it as a special acknowledgement of the help that radiology has given our professional colleagues in the diagnosis and treatment of gastro-intestinal disease that a radiologist has been invited to contribute to this symposium. I should like to express my appreciation of the privilege I have been accorded in being invited to address you in association with our distinguished visitors and lecturers Sir Gordon Gordon-Taylor and Dr. F. Avery Jones.

In the management of the complications of peptic ulceration the radiologist's task is the recognition of the pathological condition responsible for the patient's illness and the observation of its progress and the effects of treatment. It will be occupied with the diagnosis of the lesion itself,

[1] Read at a combined meeting of the Section of Medicine, the Section of Surgery and the Section of Radiology and Radiotherapy, Australasian Medical Congress (British Medical Association), Eighth Session, Melbourne, August, 1952.

its position and extent and the presence of other associated abnormalities.

Of latter years an increasing interest is being devoted to the study of reflux œsophagitis, its accompanying superficial ulceration, its usually associated hiatal herniation, and their complications of fibrosis and stenosis and in the later stages deep ulceration and even perforation.

Reflux œsophagitis is, as its name implies, due to the irritation of the œsophageal mucosa by acid pepsin gastric content, which has flowed back up the œsophagus through an incompetent cardiac sphincter. In certain instances the syndrome may be due to the presence of ectopic gastric mucosa in the œsophageal wall.

The incompetence of the cardia is now considered or explained as being due for the most part to a congenital weakness of the external sphincter formed by the right crus of the diaphragm, with which is also associated a weakness of the anchorage of the lower part of the œsophagus. This anchorage is furnished chiefly by the phreno-œsophageal ligament. This weakening of the lower œsophageal attachment offers a decreased resistance to the pull of the longitudinal œsophageal muscle fibres and results in a drawing up of the cardia and with it the fundus of the stomach, and the development of a hiatal hernia. In this the pull of the œsophagus is aided by the positive intraabdominal and the negative intrapleural pressure.

It must be emphasized, however, that incompetence and consequent regurgitation can occur apart from herniation. It may be mentioned here that regurgitation may follow operations involving the cardiac end of the stomach or be a consequence of continuous dilatation of the cardia for achalasia. Incompetence and herniation may occur at any age. They are very often associated with pregnancy and are found frequently in the middle-aged obese type of patient, the increased intraabdominal pressure in these conditions no doubt finding the weak spot at the hiatus instead of choosing the inguinal, femoral or umbilical regions.

There are two types of hiatal hernia; P. R. Allison classifies them as follows: (i) the sliding type, in which the cardia is the leading part—this type is associated with incompetence and reflux; (ii) the type in which the cardia lags behind and may even remain fixed in its normal position; this is the rolling or paraœsophageal type which is associated with œsophageal obstruction. Combinations of these two types may occur.

In the early stages the demonstration of the reflux, also of the hernia, if present, is the task of the radiologist. A. S. Johnstone, professor of radiology at the University of Leeds, who has given great attention to the radiological study of this condition, has emphasized the importance of demonstrating the reflux. The reflux may occur readily in cases of patency when the patient is simply asked to bend down into the "touch toes" attitude or assume the supine or prone position. In other cases the tilting of the table into the "head low" position, with the patient lying on the back or prone or in oblique positions, will be required to demonstrate the backflow. At other times lying on the back and flexing the neck or thighs may suffice.

The Valsalva procedure of forced expiration with the glottis closed or alternatively with the closure of the nose and mouth may be effective. Sometimes the reflux occurs on changing posture.

In the very young child regurgitation may be demonstrated by suspending the infant by the ankles. As in the adult, the confirmation of the diagnosis will be by the œsophagoscope.

In the early stage, apart from regurgitation and the demonstration of herniation, no abnormality may be shown, although some swelling of the œsophageal mucosa may be seen as well as spasm. The ulceration present is usually multiple and too superficial for radiological demonstration. It is necessary to have the assistance of the endoscopist to report on the condition of the mucosa and the extent of the involvement. It must be very strongly emphasized that the observation of reflux with or without hiatal herniation does not necessarily indicate the definite diagnosis of œsophagitis. It is, therefore, necessary to reiterate that the observation of a reflux is merely the indication for a closer investigation and, above all, for an expert and thorough endoscopic examination if the clinical picture should indicate its necessity.

A more refined technique of œsophagoscopy, the development of the flexible œsophagoscope, and a better appreciation of the contraindications to this procedure and the risks to be avoided now render œsophagoscopy a much less uncomfortable and hazardous means of investigation.

In the later development of fibrosis the radiological findings are those of narrowing deformity and non-reducibility. At times the differentiation from a stenosing type of neoplasm is not always possible, and an endoscopic biopsy taken from well within the stenosed area will be necessary. Stenosis following the swallowing of caustic liquids will also have to be considered. The outlining of the stenosed area from above and below by tilting the table is of importance in indicating the extent of the lesion.

Although inflammation, mucosal ulceration, hæmorrhage, fibrosis and stenosis are the common complications, deep ulceration and even perforation may occur. It has been pointed out by N. R. Barrett that this type of ulceration generally occurs in the herniated portion of the stomach. It may go on to perforation in some cases. Perforation may occur into the pleural or peritoneal cavities, the mediastinum, the bronchi, the lungs, the great vessels and the pericardium and heart. Such a case is the following:

> The patient was a barman, aged 50, with a long history of alcoholism. He had a duodenal ulcer, and a short œsophagus diagnosed radiologically 8 years previously when he had a severe hæmatemesis. Four months ago, following on another hæmatemesis, an ulcer crater was demonstrated radiologically in the œsophagus. He was able to go home, but returned with another hæmatemesis and died soon after admission. At autopsy a large duodenal ulcer and also chronic ulcer in the herniated portion of the stomach with a recent perforation into the left auricle were found.

At times a carcinoma may develop in the herniated stomach, as has been pointed out by Professor Smithers, of the Royal Cancer Hospital, London.

This condition of reflux œsophagitis is frequently associated with, and may be confused with, coronary sclerosis, with pyloro-duodenal ulceration and especially with cholecystitis and cholelithiasis. Carcinoma of the œsophagus and cardia will also have to be remembered in some cases. Cardiospasm will also have to be excluded in certain instances. In children reflux œsophagitis will always have to be considered in cases of suspected pyloric obstruction.

Radiology has shown that reflux from the stomach and hiatal hernia are found very frequently when sufficient time, trouble and care are given to the quest by the radiologist. But every case of reflux is not necessarily one of œsophagitis. It remains for the clinician, and especially the endoscopist, to join in the hunt for œsophagitis, hiatal herniation and ulceration of the œsophagus and their complications. In this respect radiology may be said to be acting prophylactically in the treatment of œsophagitis and œsophageal ulceration and their complications, and that endoscopy and itself are complementary to one another.

Stomach and Duodenum.

Acute perforation may be confirmed by the observance of gas under the diaphragm.

The possibility of the presence between the diaphragm and the liver of a gas-filled loop of colon, or the much less frequent interposition of a distended coil of small intestine herniated through a defect in the falciform ligament, will have to be considered from the point of view of differential diagnosis. The haustrated colonic contour and the annulated small bowel outline are informative, but if necessary a barium clysma examination in the former and a barium meal examination in the latter will be decisive. The onset

of ileus may be recognized by the demonstration of dilated bowel coils with fluid level formation.

Subphrenic abscess formation when gas-forming organisms are present is shown by a fluid level with a gas bubble above it, and a poorly moving, or more often an immobile, elevated diaphragm.

Spread through the diaphragm is recognized by pleural effusion, and involvement of the lung by the increased density of the lower pulmonary field. The abscess may localize also in the subhepatic zone or lesser sac.

Subacute or chronic perforation is revealed by the localized pocket. Perforation into the pancreas, liver and biliary passages may occur and give evidence of its presence radiologically.

Hæmorrhage.

In cases of massive hæmorrhage, owing to the indefiniteness of the clinical story it may be at times necessary to exclude such conditions as varix of the œsophagus and of the fundus, hiatal herniation or perhaps a neoplasm by means of a radiological examination. In view of the patient's condition the examination should be carried out with a minimum of disturbance and with the patient only in the prone and supine positions.

A technique has been developed along the foregoing lines in the United States of America by Hampton and others for the examination of these patients, and in their hands it has been of value.

In desperate cases and with patients who are unable to cooperate, my experience parallels that of Sir Gordon Gordon-Taylor and Dr. Avery Jones, who are of the opinion that the carrying out of barium meal investigations in such cases is unfortunately not very informative.

Changes in Stomach and Duodenum Resulting from Scarring.

Examples of changes in the stomach and duodenum resulting from scarring will be shown quickly, as their appearance is well known: the organic hour-glass stomach, the spiral rolling up of the stomach from scarring along its lesser curvature, and pyloric stenosis with its large and for the most part inert bag galvanizing itself at times into ineffectual hyperperistalsis and left with its large twenty-four hour residue. The duodenum is distorted into a bizarre shape and bulges out into a prestenotic diverticulum.

Complications following Gastro-Enterostomy and Gastrectomy.

The occurrence of acute dilatation of the stomach after operations may necessitate the assistance of the radiologist to demonstrate the position of the tip of the aspirating tube.

Stomal block or narrowing will be revealed by delay in emptying, or a gaping stoma may allow the gastric ingesta to be precipitated so rapidly into the jejunum as to be considered responsible, at least in part, for the "dumping" or the "post-gastrectomy" syndrome. This is not often seen radiologically in my experience. A too highly placed stoma may result in a poorly functioning gastro-enterostomy. Jejunal obstruction will be evidenced by dilatation of one or both loops.

Ulceration may recur in the stomach or duodenum or may develop in the stoma or in the jejunal loops. If it is of the penetrating type it can often be demonstrated radiologically, but if it is superficial it will require the gastroscope for recognition. A further complication of this latter type of ulceration is the development of a gastro-jejunocolic fistula, which is usually demonstrated by means of a barium clysma.

An uncommon complication is a retrograde jejuno-gastric intussusception. The concentric striæ are characteristic.

Conclusion.

Within the time limit it has been possible, I am afraid, to refer only sketchily to these aspects of peptic ulceration and its complications. In a symposium on such a wide subject one may fail through saying either too much about too little or too little about too much. I trust I have taken the middle course.

Acknowledgements.

I should like to acknowledge with thanks the great help I have received with lantern slides and films from the following: Dr. Marion Grossman, of University College Hospital, London; Dr. J. W. McLaren, of Saint Thomas's Hospital, London; Dr. J. Stanley Verco, of Adelaide; Dr. B. P. Anderson Stuart, of Sydney.

PRURITUS VULVÆ.[1]

By STANLEY DEVENISH MEARES, F.R.C.S. (Edinburgh), F.R.A.C.S., M.R.C.O.G.,
Sydney

PRURITUS VULVÆ indicating an itch is a symptom and not a disease. This must be borne in mind in all discussions.

In many cases, when the patient presents promptly, and the cause of the itch can be determined early, treatment can be specific and rapidly successful. Such a condition is the pruritus associated with a *Trichomonas* infection.

However, when the affection has been present for a long time, chronic changes have supervened and the ætiology remains obscure, treatment is often prolonged and unsuccessful.

In some cases histological changes in the skin have been occurring insidiously for years without disturbance of the patient, and gradually an itch appears and increases slowly, finally after years forcing the patient to seek medical aid. An example of this type of lesion is leucoplakia.

CLASSIFICATION OF PRURITUS VULVÆ.

Pruritus vulvæ may be classified under five headings, as follows:

1. Pruritus associated with a general disease: (*a*) a metabolic disturbance—for example, diabetes; (*b*) an endocrine disorder; (*c*) a toxic condition—for example, jaundice; (*d*) an allergic condition; (*e*) a blood disease—for example, leuchæmia, Hodgkin's disease; (*f*) a skin disease—for example, lichen *planus*, psoriasis, seborrhœa; (*g*) a psychic disorder.

2. Pruritus associated with a lesion of the anus or rectum.

3. Pruritus associated with genital tract lesions: (*a*) vaginal infections; (*b*) cervical and upper genital tract infections; (*c*) fistulæ—urinary or fæcal.

4. Pruritus due to a purely local condition: (a) of chemical origin, due to soaps, medicaments, contraceptives; (b) due to parasites (pediculi); (c) due to infection: (i) acute—for example, sepsis, (ii) chronic—for example, tuberculosis; (*d*) due to leucoplakia; (*e*) due to carcinoma (including Paget's disease and Bowen's disease).

5. Idiopathic pruritus.

Pruritus Associated with a General Disease.

Metabolic Disturbances.

Pruritus vulvæ may be the presenting symptom in *diabetes mellitus*. The urine of all patients with *pruritus vulvæ* must be tested for sugar. Diabetes may appear during the course of the disease when control has been inefficient. The lesion on the vulva is widespread and uniform, affecting the whole of the vulva and the skin over the upper part of the medial sides of the thighs. In colour it is dull red and beefy, and the folds of the vulva are œdematous. In some cases furunculosis may be added.

There is much discussion as to the cause of the pruritus, the commonly accepted theory being that it is *Monilia* infection encouraged by the sugar-laden urine.

[1] Read at a meeting of the New South Wales Branch of the British Medical Association on May 29, 1952.

Sometimes *Monilia* is found, but often I have searched unsuccessfully for it. The treatment is the proper control of the diabetes. At the same time I prescribe antimycotic preparations for the treatment of the vagina and vulva.

Endocrine Disorders.

Endocrine preparations, especially the œstrogens, have been exhibited for years in the treatment of all forms of pruritus, though I know of no proof that any form of primary *pruritus vulvæ* is due to a deficiency of any female sex hormone.

I remember at a British Congress of Obstetrics and Gynæcology at Belfast in 1936 that Kaufmann advocated the use of œstrin injections in the treatment of pruritus and leucoplakia, showing by means of beautifully prepared coloured slides of macroscopic lesions the improvement after treatment. I regret that I cannot excite the interest of the meeting with a similar set of slides. We all know that leucoplakia is generally a post-menopausal condition, but have all at times seen advanced leucoplakia in younger women who still menstruate and apparently are not deficient in endocrine secretions.

Some eighteen months ago I found advanced leucoplakia of the vulva proved by biopsy in a pregnant woman, aged forty-two years, and have recently treated it by excision. The lesion did not alter during pregnancy in spite of the high levels of œstrin in the blood during pregnancy.

Recently Agnes M. Stark reported a case of labour after simple vulvectomy for leucoplakia, and stated that it was very rare in pregnancy and that she could find only one reference to it in recent literature.

It is dangerous to use œstrogens, potentially carcinogenic, in a precancerous condition such as leucoplakia.

Toxic Conditions.

Generalized pruritus may occur in profound jaundice, in uræmia and with the use of certain drugs. The vulval itch is only part of this general manifestation.

Allergic Conditions.

Pruritus vulvæ may be just part of a generalized skin outbreak as a result of an allergy, or the allergic attack may be initiated by the vulva coming in contact with some substance to which the body is allergic.

Blood Diseases.

It is said that in leuchæmia and in Hodgkin's disease *pruritus vulvæ* may be present.

Skin Disease.

I shall leave a discussion of the part played by skin disease to my dermatological colleague; but first let me draw attention to a condition called by Elizabeth Hunt *lichen planus sclerosus et atrophicans*. She states that the later stages of this are indistinguishable from *leucoplakia vulvæ* and that it, too, is premalignant. Earlier she states that this condition, *lichen planus atrophicans*, is associated with other lesions on the tongue or fingers in the same patient. How a premalignant condition can have such relation to benign lesions in other parts of the body is difficult to understand. Perhaps the dermatologist can elucidate this.

Psychical Disorders.

Fear of cancer, fear of infection, fear of recurrence of pruritus may all be exciting causes of pruritus.

Pruritus Associated with Lesions of the Anus and Rectum.

Pruritus ani may spread to involve the vulva, an affection of the anus being responsible for both.

Pruritus Associated with a Genital Tract Lesion.

Vaginal Infections.

The two main vaginal infections are *Trichomonas* and *Monilia* infections. These must be searched for in all patients complaining of vulval itch.

We all have our favourite remedies for these. In general I recommend hypertonic saline douches in the treatment of *Trichomonas* infections and Bonney's blue suppositories for *Monilia* infections.

In this antibiotic age it is as well to remember that any of the antibiotics may so destroy susceptible organisms in the vagina that equilibrium is disturbed, and parasites, fungi and organisms normally held in check are free to multiply and to give rise to pruritus—for example, that due to *Monilia*.

Senile vaginitis, diagnosed by vaginal smear or biopsy, associated with a discharge, may cause some mild degree of pruritus. The senile vaginitis can be relieved by œstrogen therapy.

Cervical and Upper Genital Tract Infection.

When the pruritus is resulting from a vaginal discharge arising higher in the genital tract, it may at times be necessary to eliminate the *fons et origo*.

Fistulæ.

The possibility of the presence of fistulæ, either urinary or fæcal, opening into the genital tract by some minute opening in some hidden locality must not be forgotten.

Pruritus due to a Purely Local Condition.

Chemical Causes.

Douches, soaps, contraceptives and other medicaments may be the cause of pruritus. One's first duty is to inquire into the use of these. Frequent douching, even with water or normal saline, by washing away the normal inhabitants of the vagina, destroys the protective barrier and leaves the way open to secondary invaders.

When the patient has a part that itches she tends to think that it is because it has not been washed properly and soaps it frequently and thoroughly.

Contraceptives are a rare cause of vaginitis or of *pruritus vulvæ*.

Usually, by the time the patient suffering from *pruritus vulvæ* reaches the specialist, a large variety of chemicals have been diligently applied. It is well to inquire into this.

Parasitic Causes.

Pediculi may be found.

Infective Causes.

An acute infection of the vulva may arise *de novo* or, like the furunculosis in diabetic vulvitis, may be superimposed on an existing vulval lesion. Scratching of the vulva opens portals for the entry of infection.

Chronic infection, such as the hypertrophic form of tuberculous vulvitis, may give rise to an itch. Chronic infection of the vulva may be added to some other vulval lesion. A foreign body may be present in the vagina.

Leucoplakia.

Leucoplakia must be distinguished from vitiligo (leucodermia).

Leucoplakia is a chronic atrophic inflammatory condition of the vulval and often of the perianal regions, with the exception of the vestibule and the actual vaginal entrance. It is usually met with in women aged over forty years, but may occur earlier.

The pruritus may be intense. Later there may be pain from cracks and fissures becoming infected.

Berkely and Bonney describe the appearance in the following four stages: (i) the skin is red, swollen and excoriated, the surface dry; (ii) thickening of the skin occurs with flattening of the folds and the appearance of whitish, semi-opaque patches; (iii) the skin is diffusely white or bluish, cracked and parchment-like, with small points of ulceration; (iv) if cancer does not develop, the vulval surface becomes smooth, shiny and white, all labial and preputial folds are obliterated, and the disease becomes completely quiescent. In 30% of cases the condition becomes malignant (F. J. Browne).

R. A. Willis, referring to the chronic form of vulval dermatitis variously designated leucoplakia, leucoplakic vulvitis and *sclerodermia circumscripta*, points out that

these names do not refer to distinct diseases, but merely to different appearances or stages in the one disease, and that it is a precancerous condition. Microscopic evidence of it was present in the majority of specimens of vulval carcinoma which he had examined. He later states that in cases of leucoplakia carcinoma often develops at multiple foci, and local excision of areas of growth is likely to be followed by carcinomatous change in other parts of the diseased area. Complete vulvectomy is therefore the proper treatment of intractable leucoplakia.

Taussig found leucoplakia in 69% of his cases of carcinoma of the vulva.

Treatment of Leucoplakia.—In the treatment of leucoplakia local applications may be tried.

When the lesion is widespread and intractable, the diagnosis has been confirmed by biopsy, and ulceration and fissuring are present, the only treatment is complete simple vulvectomy. Excision must be wide or the leucoplakia will reappear at the edges of the scars.

Recently I had to excise a vulva which had been inadequately excised a few months before, the patient having been left with her pruritus and the vulval region with areas of ulceration.

When prolapse is present as well, as it was in two of my cases, vulvectomy can be combined with the Manchester operation.

Carcinoma.

In 30% of cases leucoplakia progresses to carcinoma, so all cases of advanced leucoplakia must be looked upon with suspicion. There is the danger of treating carcinoma by simple vulvectomy. When carcinoma is present, a radical vulvectomy, Bassett's operation as modified by Way, is required.

Idiopathic Pruritus Vulvæ.

In idiopathic *pruritus vulvæ*, in spite of all examinations and investigations, no cause can be found. Here in the early stages no physical signs are present, but later comes a chronic infective lesion resulting from the trauma of scratching with superadded infection. Treatment is often unsuccessful.

The Value of Biopsy of the Vulva.

If *pruritus vulvæ*, a symptom, is to cease being regarded as a disease, our diagnosis of the cause and of the true pathological process must be more exact. One way in which our knowledge can be increased is by taking biopsies of all chronic lesions of the vulva. This is easily done, does not inconvenience the patient or require her admission to hospital, and allows a histological picture to be obtained on which more exact diagnosis may be based, so that suitable treatment is able to be instituted.

Repeated biopsies are also of use in assessing the results of treatment.

Summary.

1. *Pruritus vulvæ* is discussed in general.

2. A classification is submitted.

3. The differential diagnosis of the various forms is discussed.

4. The treatment of advanced intractable leucoplakia is outlined.

PRURITUS VULVÆ.[1]

By J. Witton Flynn,
Sydney.

"Pruritus" comes from the Latin *prurire*, to itch. It is applied properly to itching which is not accompanied by any obvious changes in or disease of the skin other than those changes provoked by rubbing and/or scratching. However, it is also used loosely to denote itching even in the presence of primary cutaneous changes. It is excited in everyone by some forms of irritation, but the intensity of the subjective phenomena varies a great deal in different people. What to one is a mild discomfort is to another an intolerable irritation.

Pruritus may be local or general, and it is to a local form—that of the vulva—that I am confining my remarks this evening.

The causes of *pruritus vulvæ* are best divided into two groups, local and general.

Local Causes.

The most common of the local causes may be considered as follows.

In the first group are local irritants or sensitizers—for example, contraceptives, douches, over-cleanliness or insufficient cleanliness (smegma being allowed to accumulate), toilet paper, tight clothing *et cetera*.

The second group of local causes comprises parasites and infections—for example, *pediculosis pubis*, moniliasis, *trichomonas vaginalis* and epidermophyton (rare in Australia, Europe and Great Britain, but apparently not uncommon in America).

In the third group are diseases of the genito-urinary tract—for example, calculus, cystitis, and vaginal discharge due to lacerated cervix, cervical erosion *et cetera* (and any antiseptics used in their treatment).

The fourth group consists of local skin disease—for example, leukoplakia, *lichen sclerosus et atrophicus*, *neurodermatitis circumscripta*, and *lichen planus*.

(Here it would be wise to consider whether the four conditions just mentioned are always correctly diagnosed. It would seem that many practitioners make a diagnosis of leukoplakia simply on the presence of a white patch. Furthermore, there is need for investigation as to the precancerous nature of the different members of the group since it appears that leukoplakia is the only one with a tendency to the development of carcinoma. If this is true, then leukoplakia is the only one which requires excision. It is also worth considering (i) whether leukoplakia itself itches, and (ii) how frequently leukoplakia occurs on otherwise normal vulval skin. It may be significant that leukoplakia of the lips and mouth does not itch.)

Other causes of *pruritus vulvæ* in the fourth group are *kraurosis vulvæ* (which may cause pruritus, although more often it causes soreness), hyperhidrosis, seborrhœic dermatitis, flexural psoriasis, *condyloma acuminatum*, and an extension of the condition from *pruritus ani*.

General Causes.

Among general causes of *pruritus vulvæ* are (i) *diabetes mellitus*, (ii) neurogenic factors and habit, (iii) endocrine disturbances (for example, the menopause, pregnancy), (iv) a wide range of conditions which may cause generalized or localized pruritus—for example, (*a*) icterus, (*b*) diseases of the reticulo-endothelial system (leuchæmia), (*c*) senility, (*d*) gout, (*e*) chronic nephritis.

Treatment of Pruritus Vulvæ.

The treatment of *pruritus vulvæ* is best approached with the object of correcting the cause, and thus involves a careful investigation, to exclude the less obvious ætiological factors. For example, a glucose tolerance test may be necessary to detect mild or latent diabetes. Treatment is then conducted along the following lines.

Local irritants should be avoided.

Local infection should be eliminated; for example, shaving with the use of 10% DDT powder should be employed for *pediculosis pubis*, a 1% to 2% aqueous gentian violet solution should be used in the treatment of moniliasis, and sodium picrate insufflations should be given for *trichomonas* infections.

Genito-urinary abnormalities should be corrected.

Local skin disease should be treated; this involves one or more of the following measures.

[1] Read at a meeting of the New South Wales Branch of the British Medical Association on May 29, 1952.

Local applications may be required. In acute, weeping *pruritus vulvæ*, wet dressings of perchloride of mercury solution (1 in 5000) are applied. In less acute and mild cases, zinc cream containing 1% of solution of coal tar or 1% of phenol may be used. In chronic lichenified conditions zinc paste with crude coal tar (1%) is applied. Powders made with a talc base, and containing 5% to 10% of mild astringents such as boric acid or powdered alum, are useful in hyperhidrosis and to prevent friction.

X-ray therapy is particularly useful in long-standing cases, in which lichenification has become prominent. Fractional doses of 100*r* are given weekly for four doses, with a half-value layer of one millimetre of aluminium. The course may be repeated if necessary, but a total dose of 1200*r* should never be exceeded.

Specific measures may be required in the presence of certain local skin diseases—for example, surgical excision for leukoplakia, or the application of podophyllin for *condyloma acuminatum*.

Any associated general condition such as diabetes should be treated.

In recent years, when blood assays have been made, unexpectedly high œstrogen values in the menopause have been found together with pruritic conditions of the vulva. It seems as if the initial enthusiasm about hormone therapy is waning. In many cases it is of no value, and in many others it gives but temporary relief. In some cases it appears to bring about improvement, but the treatment must be persevered with, otherwise the pruritus returns. Some doctors are reluctant to use œstrogens locally over a long period because such treatment may cause cancer and sometimes causes uterine bleeding in menopausal women. (I should like to hear the experiences of members on this point.)

In most cases of long standing, psychological factors become important even if they were not involved in the initiation of the condition. Sedation is useful—for example, the exhibition of 0·5 grain of phenobarbital three times a day. Reassurance and discussion are necessary. Pruritus in general is highly susceptible to psychogenic influences. Every dermatologist knows that many skin affections become more itchy on excitement and calm down when the patient does so.

Pruritus may be a symptom in endogenous and reactive depressions. The sexual background of anal and vulval pruritus has frequently been analysed. Many psychoanalysts believe that the pruritic seizures and the relief of tension achieved by violent scratching are sexual or homosexual equivalents. I am unable to say in what percentage of cases this would obtain.

Occasionally severe kraurosis and leukoplakia or *pruritus vulvæ* are associated with achlorhydria. These patients are benefited by regular hydrochloric acid medication.

Surgical measures involving cutting of the various sensory nerves *et cetera* are products of inefficiency and despair. They rarely do good, and even in the few instances of relief the pruritus returns on regeneration of the nerve trunks. The same remarks apply to the injection of local anæsthetics.

Reports of Cases.

X-RAY THERAPY FOR VESICAL PAPILLOMATOSIS.

By ALAN J. M. NELSON,
Perth.

SIMPLE PAPILLOMA of the bladder can usually be successfully dealt with by cauterization through the cystoscope or, where the papilloma is larger, actual cystotomy may be necessary in order that the growth may be thoroughly destroyed. In some cases of failure the subsequent cauterization of any recurrent papillomata is sufficient to cure the condition. A certain number of cases exist in which the condition continues to recur. At each follow-up examination, at intervals of three or four months, one or two small recurrences are seen, and these are removed by diathermy. When this process continues for a matter of years it is very distressing to the patient, and the likelihood of the development of a vesical carcinoma increases. The ætiology of vesical papillomatosis is not understood, but there is a similarity to the wart which affects the skin, and one is tempted to draw an analogy between the conditions.

Verruca Vulgaris.

Verruca vulgaris has been shown to be caused by a virus infection. *Verruca vulgaris* can be cured in most cases by graduated doses of X rays to the skin. In cases of multiple warts, provided a sufficient number of warts are treated and eradicated, the condition can be cured. The mode of action of X-ray therapy appears to be the acceleration of the normal tendency to spontaneous cure from virus disease by proliferation and subsequent necrosis of virus-containing cells. During treatment those cells which are infected by the virus are devitalized. Clinically, it is found that a line of demarcation occurs between this tissue and the healthy tissue. Provided the dead wart tissue falls out or is removed, recurrence is extremely unlikely. On the other hand, if some dead wart tissue remains in the area of thickened callus, recurrence is possible, proving that it is the virus-bearing cell, not the virus itself, which is destroyed by the radiation. The suggestion was made that recurrent vesical papillomatosis might be treated along the same lines, in a series of patients under the care of Dr. F. Gill, who had been cauterizing recurrent papillomata with increasing frequency. There is very little literature on this subject, and the results of this investigation are sufficiently significant to be worth recording.

X-Ray Technique.

In all cases the position of the bladder was determined by means of a radiograph made after the introduction of a small quantity of sodium iodide. Three small areas of skin, usually circles of 7·5 centimetres' diameter, were marked out on the anterior abdominal wall and over the buttocks at the level of the bladder, and by means of a sectional diagram and the use of angulation devices X-ray beams were projected through these skin areas directly at the bladder, the volume of tissue included by the overlapping beams corresponding to the whole of the bladder. In this way it was possible to deliver to the whole bladder a dose of X rays one and a half times to twice the dose which was given to the skin on each port. The dose given to the bladder in earlier cases was over 4000*r* in four weeks, which is about two-thirds of the dose usually regarded as cancericidal. In later cases the dose given was somewhat less, because the resulting reaction of the mucosa had resulted in some discomfort in the earlier cases. The patient attended daily, two to four fields being treated each day. In general, because the fields were small, there was very little systemic upset in these cases.

Clinical Records.

CASE I.—A female patient, aged sixty years, was examined in June, 1949. This patient had reported with large papillomata five years previously. They had been fulgurated but had recurred, a cystotomy being required. Following this she required diathermy every few months for tiny incipient recurrent growths which had a very strong tendency to hæmorrhage. During the last two years the number of recurrences seen at any examination had increased, and just before the deep X-ray therapy was commenced ten match-head sized spots had been coagulated around the right lateral wall and the ureteric orifices. This patient was treated according to the technique described, except that an additional dose was delivered to the perineum and the dosage used was 5500*r* in five weeks. This patient had a little reddening and desquamation of the skin and of the vagina, which had all settled down in two months. At subsequent examinations the bladder remained clear for nearly a year, when two tiny papillomata were discovered and removed. This patient complained about

this time of occasional hæmaturia, which was found to be due to a number of small dilated veins on the posterior wall of the bladder, a telangiectasis due to a late irradiation effect. Subsequently over a period of twenty months she required cystoscopic diathermy to control small recurrences on five occasions; however, the recurrences were not so numerous as they were before the treatment.

CASE II.—A male patient, aged seventy-four years, was first examined in August, 1949. This man had appeared five years previously with hæmaturia due to several large papillomata of the bladder; subsequently there had been many recurrences, and a cystoscopic fulguration every few months had been necessary over the last few years to control the recurrences. When he was examined, a large papilloma had been removed two weeks previously. A course of treatment as described above was carried out. As a result the patient had some irritable cystitis which persisted for two to three months. Subsequent examination by the cystoscope showed that the bladder remained clear for twelve months, when a small papilloma was treated by diathermy. After this there were many recurrences, and two years after the treatment his condition was as bad as previously. One papilloma then assumed malignant properties and the patient died early in 1952.

CASE III.—A female patient, aged eighty-three years, had had intermittent cystoscopic diathermy since 1942, when she had had a small course of X-ray therapy to the bladder for a suspicious papilloma of the fundus. When she was referred by Dr. Gill in September, 1949, new papillomata were being discovered at each three-monthly examination. This patient was given a course of deep X-ray therapy with slightly larger fields, and the total dose was not so great as in earlier cases. She remained free from recurrences for twelve months, when a small papilloma was treated by diathermy. After this further recurrent papillomata appeared, requiring fulguration at intervals of about five months; but these have not been so extensive or so large as before the treatment.

CASE IV.—A male patient, aged seventy-eight years, when referred in August, 1950, had been attending every six or twelve months over the last twelve years for the diathermy of recurrent papillomata. Recently there had been two or three large and difficult recurrences. The standard X-ray technique was used, but the dose was about 3000*r* in four weeks. After this he remained free of recurrences for a year, after which a small papilloma was coagulated. Five months after this another recurrent papilloma had appeared, and it was found that the patient had developed a primary carcinoma of the colon.

CASE V.—A female patient, aged sixty-seven years, had had several severe attacks of hæmaturia over the previous two years and had fulguration of the various papillomata in the bladder on four occasions. She was treated by deep X-ray therapy in October, 1950, by the use of the same technique and with a dose of 3600*r* in three and a half weeks. After this there was cystitis which persisted for some weeks, during which time there was some dry desquamation of the skin over the treated areas. Subsequently she remained well and had no further hæmaturia.

Discussion.

Consideration of these case histories shows that after a rather strenuous course of treatment a period of remission from recurrent papillomatosis was obtained for about twelve months in all cases, but in all except one case the condition then recurred and became, after a time, almost as bad as before the treatment. In one case the condition actually went on to carcinoma of the bladder. It is evident that this method of treatment is not the answer in this rather difficult urological problem. It is doubtful whether the respite of one or two years justifies the extensive treatment employed, in view of the fact that further treatment will need to be undertaken in these cases. What that further treatment may be is, of course, a urological problem, and this series of cases is published to show that this particular line of treatment is not likely to be fruitful.

The period of remission does suggest that the analogy to the common wart may be pushed further. One might presume that as a result of X-ray damage all unhealthy cells in the bladder mucosa with malignant potential had been destroyed and shed. During the period of freedom the cystoscopic appearance of the mucosa was normal and uniform. The inference is that recurrence took place because the ætiological factor was conveyed in the urine. If this is the case, the doubtful value of all such local forms of treatment is manifest.

Acknowledgement.

Grateful acknowledgement is due to Dr. Frank Gill, at whose suggestion the investigation was made, for his cooperation in selecting these unusual cases from a large urological practice and in the carrying out of treatment.

Reviews.

Vascular Diseases in Clinical Practice. By Irving Sherwood Wright, M.D.; Second Edition; 1952. Chicago: The Year Book Publishers, Incorporated. 8½″ × 6″, pp. 552, with 116 illustrations. Price: $8.50.

"VASCULAR DISEASES IN CLINICAL PRACTICE", by Irving Sherwood Wright, is the second edition of a book published by the author. The title is self-explanatory. Wright devotes 99 pages to the study of the patient. In this he is very thorough, and gives a good account of both simple and complex methods of examination. Then he deals with *arteriosclerosis obliterans* or atherosclerosis, as it is often called. Monckeberg's sclerosis and diabetic lesions are also dealt with. A summary is given of the ætiology, morbid anatomy and treatment. Under treatment he includes rest, exercise, omission of tobacco, and the use of whisky. Whisky is a good vasodilator and is recommended for arteriosclerotic patients. With impending gangrene of a limb, one to two ounces of whisky every four hours may be helpful in keeping peripheral vessels dilated, even if the patient is kept slightly inebriated. The dose should be reduced, when the emergency has passed, to one to two ounces twice a day. Whisky may control the pain of vascular disease better than morphine. Other treatments are more conventional. In speaking of *thromboangiitis obliterans*, he strongly supports the theory that tobacco is either a cause or an aggravating factor. He describes thromboangiitis as a disease involving arteries, veins and nerves. In general, a helpful account of many vascular diseases is given, with full details and treatment. There is reference to original observations in connexion with work at the New York Hospital.

Principles of Human Physiology. By Charles Lovatt Evans, D.Sc., F.R.C.P., F.R.S., LL.D. (Birmingham), with chapters on the special senses by H. Hartridge, M.A., M.D., Sc.D., F.R.S.; Eleventh Edition; 1952. London: J. and A. Churchill, Limited. 10″ × 6½″, pp. 1222, with 709 illustrations, some in colour. Price: 52s. 6d.

IT is now forty years since the first edition of "Principles of Human Physiology" was written by the late Professor Starling and twenty-four years since Sir Charles Lovatt Evans first undertook, at its fifth edition, the revision of the book. In this, the eleventh edition, the author has once again carefully revised the text throughout, but maintained the general arrangement. It is impossible for any one man, even with such a great breadth of knowledge and experience in physiology as Sir Charles Lovatt Evans, to write with authority on every aspect of the subject. In more recent years especially, the advances in physiology and allied subjects have been too rapid to enable one person to assimilate all the changes. But, as the author points out, the book does represent in summary the principles of physiology as he understands them and as he has seen them take shape over the last forty or more years. The author has again himself revised the whole book with the exception of the chapters on special senses, which have, as in earlier editions, been written by Professor H. Hartridge.

In this edition 40 new or modified illustrations have been added. In spite of this and of additions and alterations in the text, the author has succeeded in keeping the book about the same size as the previous edition. This book has become a classic volume for all who are interested in physiology. The great amount of information incorporating experimental evidence and illustrations, together with the large number of relevant references, old and new, makes this text-book of the utmost value, not only to the undergraduate, but also to the post-graduate student of physiology.

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Basic Medical Physiology. By W. B. Youmans, Ph.H., M.D.; 1952. Chicago: The Year Book Publishers, Incorporated. 9″ × 6½″, pp. 452, with 116 text figures. Price: $7.50.

IN producing another text-book of physiology, Professor W. B. Youmans, of the University of Wisconsin, has sacrificed detailed and historical evidence and discussion of controversial questions for the sake of brevity. The whole subject of physiology is presented in only 400 pages of fairly large type. The book is written in eight sections as follows: excitation, conduction and response; circulation; respiration; digestion; nutrition and intermediary metabolism; the endocrine system; kidney and urinary tract; nervous system and senses. Each section consists of several short chapters illustrated with clearly reproduced diagrams. This concise account of physiological principles is intended to form the essential background on which to build more detailed knowledge of preclinical physiology or of clinical and pathological physiology. At the end of each section is a short list of references to other text-books, monographs and reviews which must be consulted for this more detailed knowledge.

It is doubtful whether such a very concise account of the principles of physiology is as easy for a beginner to understand as a somewhat more detailed account giving a little more explanation. No doubt some beginners in physiology will find this book useful, whereas others will prefer an introductory book which gives somewhat more experimental evidence and discussion, without which the study of physiology must be very dull.

Fractures and Orthopædic Surgery for Nurses and Physiotherapists. By Arthur Naylor, Ch.M., M.B., M.Sc. (Sheff.), F.R.C.S. (England), F.R.C.S. (Edinburgh), with a foreword by Ernest Finch, M.D., M.S. (London), F.R.C.S. (England); Third Edition; 1952. Edinburgh and London: E. and S. Livingstone, Limited. 9″ × 6″, pp. 330, with 254 illustrations. Price: 22s. 6d.

IN producing the third edition of his work "Fractures and Orthopædic Surgery for Nurses and Physiotherapists", Arthur Naylor has not only brought the subject matter up to date, but he has also substituted the modern name of physiotherapists for masseuses in the title. This work, having passed through infancy and childhood, can now be said to have matured; decked out with new illustrations and discussing modern views on newly developed therapeutic agencies like the antibiotics and cortisone, it presents a pleasing front for all nurses who are studying this specialty, as well as giving physiotherapists a complete picture of each entity for which their skilled aid is needed. Complete details of plaster technique and the use of the various orthopædic appliances are given, and the specialized treatment of different fractures is intimately described. Nurses working in the orthopædic theatre will find the details of pre-operative preparation and draping of the patients, and the photographs of the various sets of instruments most useful. Certainly this edition is a great improvement, and the book can be strongly recommended to all lecturing doctors and tutor sisters, as well as to nurses and physiotherapists.

Clinical Progress in Cardiovascular Disease. Edited by Herrman L. Blumgart, M.D.; 1952. New York: Grune and Stratton. 9″ × 6″, pp. 144, with six illustrations. Price: 4.50.

THE second of the Modern Medical Monographs series is entitled "Clinical Progress in Cardiovascular Disease" and is edited by Herrman Blumgart. It consists of a selection of papers previously published as review articles in the Clinical Progress section of the journal *Circulation*. Nearly half the monograph is devoted to a symposium on atherosclerosis, and this is the most interesting part of the book and potentially the most important. Ancel Keys draws the tentative conclusion that a "substantial measure of control of the development of atherosclerosis in many may be achieved by control of the intake of calories and of all kinds of fats with no special attention to the cholesterol intake".

The articles on "Acute Cardiac Emergencies" and "Management of Cardiac Patients in Relation to Surgery" are sound, but contain little that was not known before. Bland's paper on "Surgery for Mitral Stenosis" is already out of date and devotes too much space to procedures which have not stood the test of time. The final chapter on "Emotion and the Circulation" by Altschule derives its strength from its attention to fact and avoidance of that speculation so common amongst the psychosomatists, particularly when they speak of hypertension. It deserves to be read by all physicians.

Books Received.

[The mention of a book in this column does not imply that no review will appear in a subsequent issue.]

"Stammering Self-Cured", by A. H. C. Masterman, late R.A.R.O., The South Lancashire Regiment, with a foreword by W. Johnson Smyth, M.D., C.M. (Edinburgh); 1952. Bristol: John Wright and Sons, Limited. 7½″ × 5″, pp. 60. Price: 3s. 6d.

Described in the foreword by Dr. W. Johnson Smyth as "a small but splendid account of the most recent views relating to stammering".

"The Spread of Tumours in the Human Body", by R. A. Willis, M.D., D.Sc. (Melbourne), F.R.C.P. (London); Second Edition; 1952. London: Butterworth and Company (Publishers), Limited. Sydney: Butterworth and Company (Australia), Limited. 10″ × 7″, pp. 484, with 85 illustrations. Price: £4 4s. 6d.

The second edition of a work originally published as a monograph of the Baker Institute of Medical Research of the Alfred Hospital, Melbourne.

"Physiology of the Eye", by Arthur Linksz, M.D., F.A.C.S.; 1952. Volume II: Vision. New York: Grune and Stratton. 10″ × 7″, pp. 882, with 248 text figures. Price: $19.00.

One of three volumes. The other two are concerned with optics and physiology.

"Aids to Bacteriology", by H. W. Scott-Wilson, B.Sc., B.M., B.Ch. (Oxon.); Eighth Edition; 1952. London: Baillière, Tindall and Cox. 6¾″ × 4″, pp. 398. Price: 7s. 6d.

One of the well-known "Aids" series.

"Regeneration and Wound-Healing", by A. E. Needham, M.A., B.Sc.; 1952. London: Methuen and Company, Limited. 7″ × 4½″, pp. 160, with five text figures. Price: 8s. 6d.

A review of significant work on wound healing.

"Refresher Course for General Practitioners: Specially Commissioned Articles from the British Medical Journal: First Collection, October, 1949, to December, 1950"; 1952. London: British Medical Association. 9″ × 6″, pp. 512, with about 75 illustrations. Price: 25s.

The articles will be familiar to most of our readers.

"The Physician's Guide to Chemotherapy", by Peter N. Swift, M.R.C.P. (London); 1952. London: H. K. Lewis and Company, Limited. 8½″ × 6″, pp. 188. Price: 15s.

Designed primarily for general practitioners and hospital medical officers.

"Directory of Social Service Agencies, Sydney"; 1952. Sydney: Council of Social Service of New South Wales. 8½″ × 6″, pp. 258.

Supplies information for those who need assistance and those who administer public funds.

"Le derivazioni unipolari nell'elettrocardiografia clinica", by Giovanni Gigli; 1952. Pisa: Omnia Medica. 10″ × 7″, pp. 260, with 148 text figures. Price: L.2000.

A monograph in Italian on clinical unipolar electrocardiography.

"The Official History of Australia in the War of 1939-1945; Series V (Medical); Volume I: Clinical Problems of the War", by Allan S. Walker, M.D., F.R.A.C.P.; 1952. Canberra: Australian War Memorial. Sydney: Angus and Robertson, Limited. 9½″ × 6¼″, pp. 712, with 15 maps and diagrams and 73 photographs. Price: 35s.

The first volume of the official medical history of Australia's part in the second World War.

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The Medical Journal of Australia

SATURDAY, JANUARY 17, 1953.

All articles submitted for publication in this journal should be typed with double or treble spacing. Carbon copies should not be sent. Authors are requested to avoid the use of abbreviations and not to underline either words or phrases.

References to articles and books should be carefully checked. In a reference the following information should be given without abbreviation: surname of author, initials of author, year, full title of article, name of journal without abbreviation, volume, number of first page of the article. If a reference is made to an abstract of a paper, the name of the original journal, together with that of the journal in which the abstract has appeared, should be given with full date in each instance.

Authors who are not accustomed to preparing drawings or photographic prints for reproduction are invited to seek the advice of the Editor.

THE UNIVERSITIES OF AUSTRALIA: A NATIONAL RESPONSIBILITY.

TIME and time again during the last twenty years the universities of Australia have been discussed in these columns, and from various points of view. The last occasion was on March 22, 1952, when university development was referred to in the light of an article by Sir Douglas Copland, Vice-Chancellor of the National University, Canberra, published in the *University of Melbourne Gazette*. Among Sir Douglas Copland's views was the contention that as a nation Australia had neglected her universities, and in particular that since 1945 university development in this country had not kept pace with the requirements of a progressive and expanding economy. He discussed the financial position of universities and showed that the provision made for them was inadequate; he described the increasing number of students and insisted, as others have done, that new universities would have to be established; and he named certain "gaps" in the structure of those already in existence.

Sir Douglas Copland's article, however, appears to have been a prelude or an overture to a more serious communication which comes from the Australian Vice-Chancellor's Committee.[1] Notice should be taken of this pamphlet for two reasons. The first is the urgency of the subject; the second is the fact that the Vice-Chancellors make the matter a national concern. The pamphlet is divided into two parts. There is a six-page introduction and eighteen pages are devoted to the pamphlet proper. The Vice-Chancellors, of whom Professor S. H. Roberts, Vice-Chancellor of the University of Sydney, is chairman, are better qualified than anyone else to speak about the needs of Australian universities; indeed, their whole lives are spent in the universities' *ménage*. Their object is first to state the case for immediate increased assistance to universities, not only from the governments of the States and the Commonwealth, but also from industry and private resources, and secondly, to draw attention to the pressing need to make plans now to meet university requirements of the future. Discussing the role of universities in a modern community, the Vice-Chancellors refer to their responsibility for the training of specialists necessary to the community—doctors, scientists, engineers, architects, teachers, lawyers, economists and so on. They point out that if universities are neglected and teaching standards are not maintained, the standard of university graduates will inevitably deteriorate, and sooner or later the living standards of the community. This is what we may call the utilitarian aspect of university functions. The Vice-Chancellors also refer to the value of research and to the pursuit of knowledge for its own sake. They point out that research activity has been restricted in Australian universities because of the heavy teaching load imposed on staffs by the increasing number of students, and also by the lack of adequate funds for research. They point out what the general members of the community fail to realize, that if university staffs are to retain the inspiration and vigour necessary to a high teaching standard, they must have a reasonable opportunity to undertake research in their own subjects—in other words, they cannot be teachers unless at the same time they are learners. The Vice-Chancellors try to impress these facts on the community by referring to the value of research connected with industry. People realize the value of research when they hear that some national scourge such as the prickly pear has been deprived of its malignancy, or that some industrial hazard may be set at nought. They cannot so readily understand why it is important that men and women should be trained to think, that they should enlarge their fields of vision, and that they should be able to develop new ideas which could be put to useful purposes. It is true that the nature of universities is changing. The Vice-Chancellors name two factors. The first of these is the impact of technology. This is a factor of the modern day which has to be considered, but ways and means have to be found of binding the study of technology with what we may term culture of the mind. The Vice-Chancellors think that the way in which the humanities are being "crowded out" in favour of more practical disciplines could, if permitted to continue, undermine the whole concept of university education "which has its very foundation in a liberal, rather than a specialized training". The second factor is the increased extension of the opportunities of young men and women to obtain a university education. The universities are indeed now regarded as "an open door of opportunity for all able young people", and less of a preserve for the privileged few. This will surely receive the general approval of the community, but the community must not be foolish enough to think that the universities can meet the increased demands upon them unless the resources necessary to the task are provided. The story of the overcrowding at universities is well known to readers of this journal and need not be repeated. At the same time, it may be worth while to point out that the student enrolments for 1945 at Australian universities were 15,586; in 1950 they were 30,630; the predicted enrolments for 1955 are 31,300, and for 1960 and 1965 they are 39,100 and 47,500 respectively. It takes some years to create new universities and to make them function, and we must realize that even if an extensive building programme was put in hand at once, the increase on the

[1] "Crisis in the Finances and Development of the Australian Universities", prepared by the Australian Vice-Chancellors' Committee. 9½" × 6", pp. 24. Price: 2d., post free. (Obtainable from the Secretary of the Committee, National University, Canberra.)

debit side would not be wiped out during the next ten years. We thus see that the Vice-Chancellors tell a sad story about undergraduate education. When we turn to post-graduate work the state of affairs is not much better. Many of the universities are in need of modern equipment to replace what is "really obsolete". It should be possible for graduates to complete their training in this country, and yet we are told that organizations such as the Commonwealth Scientific and Industrial Research Organization have to send university graduates overseas to complete their training. To some extent the newly established Australian National University is thought likely to help in the restoration of balance between the undergraduate and post-graduate training in Australia, but we may be sure that this restoration will not be attained without action by the State universities.

The revenue of Australian universities is derived from students' fees, government grants and endowment income. Only a small portion of the cost of university courses is met by students' fees. In 1939, students' fees comprised only 26·4% of the total university income, while in 1951 it was estimated that this had fallen to 18·6%. Universities have increased their fees. The University of Melbourne has increased its fees since 1951 by 87%; the University of Queensland has increased its fees since 1951 in most cases by 100%, and since the end of the war the University of Sydney has been forced to increase fees by no less than 181%. No further worthwhile increases in students' fees can be made. Income from private endowment has fallen. In 1939 17·4% of university revenue came from this source, but now only about 5% of revenue is so derived. Though the Vice-Chancellors remark, perhaps hopefully, that the state of affairs does not indicate that there is no opportunity for private individuals to help universities, most assistance will have to be forthcoming from Commonwealth or State government grants. We read that the enactment of the *States' Grants (Universities) Act*, 1951, has done much to improve the financial position of the universities. Under this Act, the Commonwealth is providing grants to the States for university purposes, of from £803,000 to £1,103,000 *per annum* for three years, depending upon the amounts by which the State governments are prepared to raise their own grants to universities. Unfortunately, the universities are finding that their income from the Commonwealth is declining as the number of students under the Commonwealth Reconstruction Training Scheme falls. It is shown that in 1951 the total increased income of universities was approximately £1,150,000, made up as follows: an increase in State grants of £475,000, an increased income from students' fees of £225,000, and an increase in Commonwealth grants of £450,000. Unfortunately, "even this substantial improvement in the finances of the universities does not place the universities on a sound footing or enable them to plan the development necessary with any confidence". An interesting comparison is made of the amount of money spent on the current running costs of universities in different countries. In the United Kingdom, the amount (in Australian currency) spent per head of population is 12s., in the United States it is £3, in Canada it is £1 5s., in Australia it is 11s. The percentage of the national income spent on education in these four countries is 0·21, 1·36, 0·31 and 0·15.

The final question is what is to be done. The Vice-Chancellors state that the first essential is the "eradication of the weaknesses existing in the present university institutions". It is not quite clear to what this refers. Presumably it has reference to overcrowding, to the lack of equipment, to the increase of post-graduate facilities and so on. It is suggested that the already constituted Commonwealth Committee should proceed immediately to review the level of Commonwealth assistance to universities and to pay special regard to the effect of inflation on the funds provided. It is also stated that action should then be taken to ensure that sufficient resources are available to enable the universities to establish a reasonable ratio of staff to students. The other point mentioned is that for a plan for long-term development. The Vice-Chancellors seem a little diffident and remark that already the universities of Sydney and Melbourne "may be" beyond the optimum size for a university. Surely they have an opinion on this, since they observe that these universities are already almost twice the size of the large provincial universities of the United Kingdom. In a discussion on long-term development, the Vice-Chancellors are altogether too meek and mild. They should take off their gloves and do a little straight hitting. They do make one good suggestion when they remark that future development of university facilities should be on a national rather than on a regional basis. All this is to be left to a committee. The deliberations of a committee offer a convenient method for the handling of difficult situations. Sometimes success follows; more often there is failure. It is hard to know what to suggest. One is tempted to think that the individual universities might give the Vice-Chancellors authority to become a committee of action and make the necessary approach to Federal and State governments. Whether this is done or not, everyone in the community should be aware of the facts, and medical practitioners as university graduates should be able to do something in the field of propaganda.

Current Comment.

ISOTOPES IN MEDICINE.

WE are accustomed to find admirable summaries in the *British Medical Bulletin*, but it may be doubted if ever a better digest was presented than that dealing with isotopes in medicine recently issued.[1] Were it not that progress in this branch of medical science is so rapid, filling in gaps and following wise suggestions, one would recommend that the treatment here given should be expanded into book form. The first article, dealing with principles of assay and standardization, is highly technical and will appeal chiefly to physicists. That on counters and ion chambers for the detection and measurement of radioactive isotopes is less recondite and will be found useful by those who employ this method of investigation. Unless he has followed the more recent work on isotopes, the ordinary medical reader will be surprised at the number of applications of this technique to very practical matters in physiology, pathology, medicine and surgery. He already knows that substances can be followed from food to tissues, such as alimentary iron to red cells and bone marrow, that the stages of chemical synthesis can be revealed, and further that radioactive matter can be used therapeutic-

[1] *British Medical Bulletin*, Volume VIII, Numbers 2 and 3, 1952.

ally, especially when there is accumulation of some element in a particular organ, such as iodine in the thyroid; but the range of useful employment in all branches of basic and clinical medicine will assuredly arouse wonder. He may be curious to know how radioactive technique can be of service in plastic surgery, and here he will learn that a very practical contribution can be proffered, for the circulation in a pedicle can be assessed with accuracy. What happens when penicillin is given by mouth? We now discover that only a fraction, one-quarter to one-half, is absorbed from the intestine, the rest being chemically degraded and excreted. The fat in milk is shown to be produced by endogenous metabolism in the mammary gland and not by absorption of fatty matter from the blood, the proof being that radioactive acetate, intravenously injected, gives butter fat with several hundred times the radioactivity of the fats in blood plasma. In immunity problems the detection of radioactivity is immeasurably more delicate than any chemical analysis and approaches the reactivity of the living animal. Testing of thyroid function, treatment of bladder carcinoma and measurement of blood volume are amongst the investigations described. But perhaps the most startling contribution is that a new chapter in hygiene has begun and has its future outlined. Both patient and operator must be safeguarded against noxious dosage or exposure. The wearing of protective coats and gloves, the handling of contaminated articles, how to cleanse the hands if contaminated, the disposal of radioactive waste, liquid, solid and gaseous, are all described. In connexion with the last named it is suggested that the stack emitting radioactive vapours should be 10 metres higher than the top of any building 200 metres down wind. Here certainly is something new in health regulations. The final chapter in this symposium is a very practical one and tells how the medically important isotopes can be produced and supplied. "The Isotope Division at Harwell alone is making between 800 and 1000 shipments of radioisotopes each month to 28 countries." This must be interesting reading for Australians.

BARBITURATE POISONING.

A GREAT deal of attention has been paid to acute poisoning by barbiturate drugs, and its importance is emphasized by the not uncommon signs of subacute or chronic intoxication. The ready accessibility of these drugs is notorious also, for in spite of restrictive conditions imposed by legislation, many domestic drug cupboards contain potentially dangerous quantities. Faced with a case of acute poisoning, the physician usually does not know what dose has been taken; but that is less important than a just estimate of severity of the symptoms, and treatment really consists of the routine of elimination, if possible, by gastric lavage, and the administration of analeptic drugs. Most, if not all, of the latter are in themselves toxic and require great care. All these matters are really common knowledge, but various authors have recommended a simple scheme of classification, so that the appropriate treatment may be logically applied. Charles E. Reed, Marshall F. Driggs and Charles C. Foote have attempted to fill this gap with a scheme which they have put to the practical test.[1] In a little over eleven years they collected 300 cases of acute barbiturate intoxication, and carried out treatment in the last 91 of these according to a logical scheme. This scheme depends on an assessment of the severity of the symptom complex, which is itself based on the depression of the nervous system, the respiration and the circulation such as is familiar in the various stages of anæsthesia. Five groups are recognized in this scheme. In Group O the patient is asleep but can be roused and will answer questions, take drinks *et cetera*. In Group I the patient is comatose, though with no circulatory embarrassment or reflex disturbance, and will withdraw from painful stimuli. In Group II no response to such stimuli is seen, the reflex disturbance is slight, but there is no depression of the circulation or respiration. In Group III most of the reflexes are in abeyance, but there is still no depression of circulation or respiration. Patients in Group IV have, in addition to practically absent reflexes, respiratory depression with cyanosis, or circulatory failure, or both. It will be seen that the intermediate groups depend for their recognition on the state of the reflexes. The first 209 cases were studied in the light of this scheme, and attempts were made to correlate the dose taken with the fatalities, the length and depth of coma, and the importance of signs such as cardiac arrhythmia. Naturally no useful coordination can be made in most instances. One practical point is stressed, that no attempt should be made to estimate the grouping of an individual patient until an adequate airway has been established, as obstructive anoxia will cause areflexia and cyanosis. Cardiac arrhythmias were studied with some interest. Reed, Driggs and Foote found that there seemed to be a definite relationship between death from cardiac anomalies and the dosage of drugs such as caffeine and amphetamine. As the result of this study they conclude that the safe upper limit is six grammes of caffeine and 300 milligrammes of amphetamine in twenty-four hours, since there is a possibility of myocardial disturbance with fatal arrhythmia prejudicing a patient's chance of recovery. Arrhythmia was in fact the commonest single determining cause of death, followed by pneumonia and depression of the central nervous system. Hyperthermia was also observed in a few patients. After carrying out this study, Reed, Driggs and Foote drew up a programme of therapy based on their classification of the depth and severity of coma. Of course they point out the importance of all the routine observations on circulation, respiration and reflex activity in addition. The details of handling need not be given here, as they follow standard practice. The following generalizations are useful. Patients within the Groups O and I categories should be observed lest they show signs of slipping into deeper coma, but they should recover without special treatment. Group II patients should also recover, but may be aided by analeptics and thereby have fewer complications. The treatment recommended here is to give caffeine and sodium benzoate up to 0·5 gramme every two hours, care being taken not to exceed six grammes in twenty-four hours, and amphetamine in addition or separately, 25 milligrammes every two hours, but not more than 300 milligrammes in twenty-four hours. It is with Group III that they have found analeptics indispensable, as many of these patients will die without them. If caffeine and amphetamine are not effective in restoring reflex activity in about twenty minutes picrotoxin is recommended. It is pointed out that this drug acts on the spinal and medullary centres, its action is usually quick and its effect passes off quickly. Sometimes, however, it shows delayed action. The dangers of this drug are well known. Reed, Driggs and Foote use it to restore reflex activity, not to abolish coma, and take twitching as a warning sign. They find that it may be used in doses of one millilitre, increasing every twenty or thirty minutes, remembering that overdosage will occur if twitching, the premonitory sign of convulsive seizures, is disregarded. A sound knowledge of the pharmacology of the drug is essential for safety. For the Group IV cases higher dosage may be used. Oxygen requires caution in its use. Although it may be used helpfully in all other grades, when there is already respiratory depression it is risky to remove the natural stimulus to breathing, and carbon dioxide will still further narcotize the medullary centre; therefore artificial respiration of some kind is necessary. If circulatory shock exists, transfusion should be considered. The writers describe the methods and drugs that they have used personally; "Metrazol", for example, they mention, but have not used. They state that the mortality rate in their last 91 cases in which their ordered method was used was 5·5%, whereas it was 13·4% in their first 209 cases. It is rather horrifying to read of 300 patients with acute barbiturate poisoning being admitted to one hospital in eleven years, but it shows that the emergency is one which may confront any practitioner, and a logical approach should improve the standard of treatment.

[1] *Annals of Internal Medicine*, August, 1952.

Abstracts from Medical Literature.

PHYSIOLOGY.

Survival of Transfused Red Blood Cells.

P. L. MOLLISON, H. A. SLOVITER AND H. CHAPLIN, JUNIOR (*The Lancet*, September 13, 1952) state that human red blood cells stored in 15% glycerol solution at −79° C. for six months are capable of normal survival after transfusion. When they are stored at −15° C. for about eighty days, only a small proportion of the cells can be recovered after thawing; however, these also have a normal post-transfusion survival. The implications of these observations are discussed.

Experimental Cooling of the Blood-Stream.

E. J. DELORME (*The Lancet*, November 8, 1952) describes a method for investigation of the physiological effects of cold in which the animal is cooled by circulating its blood through a refrigerating system outside the body. He states that cooling of the blood-stream appears to be a practicable method for lowering the respiratory activity of the cell without depressing the function of essential tissues below a level compatible with life. At a body temperature of 25° C. dogs can survive drastic and prolonged reductions in blood volume as well as suspension of blood flow, both local and systemic. Cooling brings about what may be termed temporary degradation of the more delicate higher organism to the level of the more resistant lower organism, and in so doing enables it to withstand otherwise lethal ischæmia.

Spread of Excitation through the Ventricular Myocardium.

H. B. BURCHELL, H. E. ESSEX AND R. D. PRUITT (*Circulation*, August, 1952) have studied the spread of the excitatory process in the ventricular septum of the dog's heart, using the isolated organ perfused by the Langendorff technique. They found that it was possible to remove large portions of the free wall of the right and left ventricles and explore the septal surfaces under direct vision. They state that the mean excitatory pathway in the muscular mass of the dog's ventricular system was demonstrated to be from apex to base. The left side of the septum is activated before the right side, and the apical part of the right side is activated before its base.

The Influence of Airplane Noise on Auditory Thresholds.

BEN H. SENTURIA (*Annals of Otology, Rhinology and Laryngology*, June, 1952) states that there has been a great deal of controversy regarding the degree and permanence of hearing impairment as a result of exposure to airplane noise. A group of 100 cadets commencing primary flying training were checked clinically and audiometrically and were subsequently retested at the completion of the primary course in open cockpit planes with a noise level of 110 to 120 decibels. Later tests also were made after completion of a further course of basic training and finally after the advanced training course. A larger percentage of the men showed temporary hearing losses following periods of exposure to aircraft noise. These losses tended to involve the frequencies 1000 to 6000 cycles per second. On the whole these hearing losses were completely recovered from after twenty-four hours of freedom from exposure to aircraft noise. In striking contrast were a group who had not been rested during the later part of advanced training. These showed mean hearing losses which were widespread involving the frequencies 1024 to 5792 cycles per second. In the more vulnerable 2896 to 5792 cycles per second region, recovery of hearing is delayed.

BIOCHEMISTRY.

Alloxan Diabetes.

J. H. COPENHAVER *et alii* (*Archives of Biochemistry and Biophysics*, December, 1951) have compared the activities of anaerobic glycolysis, the tricarboxylic acid cycle, pyruvate oxidation, succinic dehydrogenase, glutamic dehydrogenase and transaminase in tissue of normal and alloxan diabetic rats. Of the above enzyme systems, all except glutamic dehydrogenase were found to be increased significantly in the livers of alloxan-diabetic rats. The succinic dehydrogenase activities of *corpora lutea* of lactation, brain and kidney of diabetic rats were normal. Likewise, the activity of transaminase in kidney and heart was unaltered in the diabetic animal.

Folic Acid.

M. SILVERMAN *et alii* (*The Journal of Biological Chemistry*, February, 1952) have shown that in folic acid deficiency induced in the rat by succinylsulphathiazole, a heat-labile and alkali-labile compound appears in the urine. The compound contained at least 50% of glutamic acid.

Glycogen.

L. COTÉ *et alii* (*Archives of Biochemistry and Biophysics*, December, 1951) have shown that anise oil, anethole and several related compounds inhibit the deposition of glycogen in the livers of fasted adrenalectomized rats by adrenal cortical extract. This effect is reversed by increased levels of adrenal extract. Anethole blocks the effect of adrenal cortical hormones in reducing insulin sensitivity of adrenalectomized rats. Adrenal hypertrophy of unilaterally adrenalectomized rats is increased by anethole. It is postulated that these compounds compete for an active carbohydrate intermediate formed during glycogenesis. The authors state that β-glucuronidase may be involved in this phenomenon.

Cobalamin.

U. J. LEWIS *et alii* (*The Journal of Biological Chemistry*, February, 1952) have isolated a vitamin B_{12}-active material present in the fæcal matter of rats. They state that the compound has growth-promoting properties for *Lactobacillus leichmannii*, but is inactive in this respect for the rat. The growth response of the chick to the material is comparable to that of crystalline vitamin B_{12}. An increased production of this factor in the intestinal tract of rats was noted when inorganic cobalt was fed to the rats. The material is not stored in the liver of the rat to any great extent. The absorption spectrum of the material is not identical with that of vitamin B_{12}.

G. H. SPRAY (*The Biochemical Journal*, March, 1952) has shown that the factor in normal human gastric juice which combines with vitamin B_{12} so that the latter is not available as a growth factor for microorganisms is more stable to heat in neutral solution than in acid solution. The heating of mixtures of gastric juice and vitamin B_{12} under conditions which left a considerable part of the vitamin in a form unavailable to bacteria, destroyed the ability of the juice to potentiate the action of orally administered vitamin B_{12} in a patient with untreated pernicious anæmia. These results suggest that the vitamin B_{12} binding factor is not identical with the intrinsic factor of Castle.

Plasma Protein.

I. A. ABDOU *et alii* (*The Journal of Biological Chemistry*, January, 1952) have shown that labelled plasma proteins pass rapidly from either the circulation or the peritoneal cavity of anæsthetized rats into the thoracic duct lymph. In either case maximum labelling of the lymph protein occurs in three or four hours. After the labelled protein is given orally, little or none appears in the lymph. The results support the concept of a continual circulation of protein from blood and extracellular fluid into the lymph and back again into the blood.

Fat Absorption.

R. REISER *et alii* (*The Journal of Biological Chemistry*, January, 1952) have studied the mechanism of fat absorption using synthetic triglycerides in which the glycerol was labelled with C^{14} and the fatty acids with conjugated double bonds. Approximately 25% to 45% of the ingested glycerides was completely hydrolysed during absorption. The remaining 55% to 75% of the ingested glycerides was hydrolised to monoglycerides, but followed an independent metabolic pathway. Half the lymph phospholipide formed from ingested fat utilized the hydrolysed fatty acids and endogenous glycerol. The other half was synthesized from absorbed glycerides.

Sulphur.

F. BINKLEY *et alii* (*The Journal of Biological Chemistry*, January, 1952) have examined aged dialysed preparations of the enzymes responsible for the transfer of the sulphur of homocystine to the carbon chain of serine with the formation of cysteine. These preparations were greatly activated by the addition of minute amounts of pyridoxal phosphate. Liver tissue of rats maintained on a diet deficient in respect to pyridoxine was found to have significantly less activity than liver tissue of control animals. The

preparations from the deficient animals were activated to near control levels by the addition of pyridoxal phosphate.

Insulin.

N. HAUGAARD AND J. B. MARSH (*The Journal of Biological Chemistry*, January, 1952) have studied the metabolism of retroperitoneal adipose tissue from rats fasted for twenty-four hours. They found that addition of insulin *in vitro* increases the oxygen uptake of this tissue in the presence of glucose, lactate or acetate. Insulin has been shown to combine chemically with this tissue in a manner similar to that with rat diaphragm. This finding strengthens the concept that combination of insulin with tissue is a prerequisite for insulin action.

MEDICINE.

Hereditary and Idiopathic Clubbing of the Fingers.

M. C. MCCORD AND H. L. HYMAN (*Diseases of the Chest*, September, 1952) describe two cases of hereditary clubbing and two cases of idiopathic clubbing of the digits. The authors consider that clubbing does not necessarily indicate the presence of a visceral lesion.

Poisoning by Chlordane.

P. LENSKY AND H. L. EVANS (*The Journal of the American Medical Association*, August 9, 1952) describe a case of poisoning due to the new popular commercial insecticide, chlordane, a chlorinated hydrocarbon, which may enter the body through the intact skin, by inhalation of dusts or sprays or by ingestion. A child, aged fifteen months, drank a small quantity of a chlordane suspension. She spat some out and within two or three minutes was made to vomit by the administration of a solution of sodium bicarbonate. Three hours later a generalized tremor developed, and the child became irritable and ataxic. Convulsions ensued with sardonic grin and opisthotonos. These were controlled with a barbiturate drug, and the stomach was washed out with magnesium sulphate, chlordane being recovered. Later there was respiratory depression which could have been due to the anticonvulsant drug. After sixteen hours the child roused and in two or three days lost the irritability and ataxia.

Venous Thrombosis.

W. E. LORING (*The American Journal of Medicine*, April, 1952) discusses venous thrombosis in the upper extremities as a complication of myocardial failure. He states that in myocardial failure a large proportion of thromboses occur in the lower limbs; about one in fifty occur in the upper limbs. Seventy-eight cases of the latter type were collected from the literature; most occurred in middle age. All types of heart disease were involved, rheumatic, hypertensive, coronary, syphilitic and so on. The condition occurs more frequently in females, and often after several months or years of heart failure. The thrombi occurred on the left side most frequently, and in the arm seven times as often as in the neck. The subclavian and jugular veins were affected three times as often as the superior *vena cava*. The onset was rapid or slow. Pain and œdema of the arm, neck or face were noted in many cases. Tenderness and thickening of veins were common. Pulmonary infarction was a complication in 36 cases. It was fatal in three cases. The prognosis of venous thrombosis in the arms was bad.

Leuchæmia in Doctors.

S. PELLER AND P. PICK (*The American Journal of the Medical Sciences*, August, 1952) conclude that physicians as a body, and not only radiologists and dermatologists, develop leuchæmia much more commonly than the general population. The increased mortality is apparent even in the twenty-five to twenty-nine years age group and is about three times normal in all age groups. They attribute the increasing incidence among physicians to the increasing use being made of fluoroscopy and to inadequate protection.

ACTH and Wasp Stings.

E. STEWART JAMES AND W. G. WALKER (*The Canadian Medical Association Journal*, July, 1952) gave ACTH to an eighteen-months-old child who had received 477 wasp stings. They suggest that the child's recovery may have been aided by this drug and refer to reports of success with its use in cases of snake and spider bite.

Fractures in Cortisone-Treated Patients.

F. DEMARTINI *et alii* (*The Journal of the American Medical Association*, June 21, 1952) report fractures sustained by five patients receiving cortisone therapy for rheumatoid arthritis for periods of two to twenty months. All the patients were women past the menopause, in whom osteoporosis usually accompanies chronic arthritis. However, as cortisone gives rise to osteoporosis, it is possible that the cortisone may have caused the fractures, owing to softening and weakening of bone, in these patients. All the patients required 500 milligrammes of cortisone weekly to obtain a minimal palliative effect. All the patients had been in bed for some time, and this may have accounted for some of the osteoporosis observed. One patient fractured her femur and suffered a compression fracture of the sixth and eighth thoracic vertebræ after a convulsion. Another had collapse of the twelfth and fourteenth thoracic vertebræ with marked osteoporosis of the spine. It is not certain that cortisone was the whole cause of the osteoporosis and the fractures.

Acute Pericarditis of Unknown Origin.

D. H. DAVIES (*British Heart Journal*, July, 1952) describes five cases of acute pericarditis of unknown origin and gives an account of the condition and the features which distinguish it from acute myocardial infarction, rheumatic and tuberculous pericarditis and pleurisy. He states that the importance of recognizing this benign complaint lies in the avoidance of the prolonged invalidism and gloomy prognosis attached to the other conditions. Its main features are pain, pericardial friction, often pleurisy, the electrocardiographic changes of pericarditis, fever, and complete recovery usually within two months. There is no special treatment.

Quinidine in Myocardial Infarction.

F. B. CUTTS AND B. RAPOPORT (*The New England Journal of Medicine*, July, 1952) do not support the routine administration of quinidine. They state that in 100 cases in which treatment was carried out with quinidine (three to nine grammes eight-hourly) there were as many deaths as in 100 control cases.

The Venous Pressure in Pneumoperitoneum Treatment.

G. J. BECK *et alii* (*Diseases of the Chest*, August, 1952) have found estimation of the venous pressure to provide valuable prognostic information in pneumoperitoneum treatment. A fall in the venous pressure after the establishment of a pneumoperitoneum indicates that the treatment is likely to be beneficial; it is thought that an increase in diaphragmatic excursion is probably the cause of the fall in venous pressure. A rise in venous pressure is a bad sign and is probably due to transmission of the rise in the intraperitoneal pressure through a fixed atrophic diaphragm to the thoracic cavity; the return of blood to the right side of the heart is thus impeded and respiratory embarrassment results.

Intestinal Tuberculosis.

F. PIECHAUD *et alii* (*Diseases of the Chest*, July, 1952) report favourable results from the use of streptomycin in the treatment of intestinal tuberculosis. Diarrhœa is usually stopped in a few days. Thiosemicarbazones are less effective than streptomycin. Para-aminosalicylic acid is sometimes badly tolerated.

Splenectomy in Leuchæmia.

J. H. FISHER, C. STUART WELCH AND WILLIAM DAMESHEK (*The New England Journal of Medicine*, March, 1952) report good results from splenectomy in 18 selected cases of leuchæmia and leucosarcoma. These cases comprised 5% of all those in which they performed splenectomy during ten years. They state that the main indication for this treatment is "hypersplenism", as shown by hæmolytic anæmia or various types of cytopenia, and only rarely discomfort due to the largeness of the spleen. They emphasize that the operation is not a treatment of leuchæmia but only of the occasional case in which splenic malfunction is present.

Angina Pectoris and Œsophageal Hiatus Hernia.

F. R. NUZUM (*The Journal of the American Medical Association*, April, 1952) draws attention to the similarity between the symptoms of *angina pectoris* and those of hiatus hernia, and emphasizes the importance of distinguishing between these two conditions. Among 100 patients suffering from *angina pectoris* he found 25 (25%) with hernia, and six of these were relieved by medical treatment. Among 975 patients investigated for gastro-intestinal troubles he found only 12% with hernia.

Special Articles for the Clinician.

(CONTRIBUTED BY REQUEST.)

XLVIII.

BRONCHIECTASIS IN CHILDHOOD.

"I've that within for which there are no plasters."

BRONCHIECTASIS is a chronic disease of childhood whose onset is frequently insidious, and in many cases it is the anxiety of the parent rather than the appearance of the child that makes the medical attendant aware of the serious nature of the condition and prompts the necessary investigations. The text-book description of a child with constant cough and large quantities of foul sputum, stunted in growth, with a considerable degree of cyanosis and clubbing of the extremities is that of an advanced stage of the disease which is now, fortunately, rarely seen. The statement, "When once the classical picture of bronchiectasis has developed the condition should be regarded as grave, and likely to progress to a fatal termination", is no longer true.

As in other chronic diseases it is essential that the practitioner should have an understanding of the underlying processes and what may be expected of treatment, so that his interest and enthusiasm will be maintained; for the parents are quick to realize a lack of interest and will seek new advice if their fears are treated lightly and they do not see the benefits of treatment in a short time.

The child suffering from bronchiectasis cannot be considered the special problem of a general practitioner, physician or surgeon, radiologist or bronchoscopist, but in the ideal management all should play their part. This arrangement is not difficult to achieve in hospital practice, and most large hospitals have special departments or clinics for the study and investigation of the problems involved, but in private practice the responsibility will inevitably fall on the general practitioner in the first place to recognize the disease in its early stages or to suspect its presence and then to direct the patient for the proper investigation so that treatment may be instituted.

Pathology.

Although there may be some points in the production of bronchiectasis that are not fully understood, sufficient is known to provide a working basis for the management of the child. The two factors principally concerned are atelectasis and infection. It is well known that bronchiectasis will frequently follow an attack of pertussis or measles, but it is not so well known that a much commoner method of onset is an insidious development following repeated attacks of bronchitis, and that the initial infection in over 80% of the cases occurs before the age of two years.

In these infections and also in the bronchitis which is an almost constant accompaniment of pink disease, areas of collapse frequently occur. These may be small or may be demonstrated radiologically and are caused by thick tenacious mucus obstructing a bronchiole. This is a common event in a child, in whom the bronchial tree is of small size and less rigid than in later life. In most cases this mucus will be expelled by coughing, but if it is retained atelectasis results and bronchiectasis may follow.

The other factor concerned is weakening of the bronchial wall by the inflammatory process, which may result in destruction of the normal structures and their replacement by fibrous tissue.

If atelectasis is present the weakened bronchial wall is subjected to the dilating effect of the negative intrathoracic pressure directly transmitted to the bronchus through the collapsed lung, whereas normally it is "cushioned" by the air-containing tissue surrounding the bronchus. The end result of this process is dilatation of the smaller bronchi.

Atelectasis by itself does not cause bronchiectasis. A portion or the whole of a lung may remain collapsed for many months by the pressure of a tension cyst in another part of the thorax. When this cyst is removed the collapsed lung will expand and remain expanded without developing bronchiectasis; so that the infective factor remains by far the most important.

Clinical Features.

The clinical picture of a child with advanced bronchiectasis is not difficult to recognize. It has already been suggested. Before this complete picture is established there will be a period of some years during which the child suffers from repeated attacks of infection of the respiratory tract usually diagnosed as pneumonia. During these attacks increased amounts of sputum are produced, and adventitious sounds may be heard over both lungs. It is the recurring pyrexial attacks and the persistence of cough that lead to the suspicion that bronchiectasis may be in the process of development. When an acute attack has subsided, persistent râles will be found at the affected areas, usually at the bases or in the axillæ if the lingula or middle lobe is affected. The râles will be accentuated or may be heard only after a cough followed by a deep breath. The percussion note may be impaired on the affected side, but sometimes is hyperresonant if there is much compensatory emphysema.

In such a case it will often be found in retrospect that the child suffered from frequent attacks of bronchitis commencing during the first two years of life, and that after one of these attacks the cough did not clear up. Further pyrexial attacks occurred and the sputum increased in amount until the complete picture was established.

The other method of onset is following an attack of one of the specific infections in childhood, especially whooping-cough or measles. In these cases convalescence is not satisfactory, and the cough never really clears up. It is not the severity of the attack which is the determining factor in the development of bronchiectasis, but the occurrence of pulmonary collapse which persists. The lower lobes of the lungs are more frequently affected by collapse, and this corresponds to the usual site of bronchiectasis. As it is more difficult for plugs of mucus to be expelled by coughing against the force of gravity, the anatomical arrangement by which the lower lobes are dependent is probably an important factor favouring their more frequent involvement.

Sinus Infection.

The majority of children with bronchiectasis suffer from infection of the maxillary antrum, but the common conception is that this is not the cause of the bronchiectasis, and that it occurred at the same time as the initiating infection. There is a close relation between the two conditions without either being really dependent on the other. It is important, however, that this condition should not be overlooked when treatment is being considered.

Investigation.

When cough persists for an unduly long period after a respiratory infection, the suspicion will be aroused that bronchiectasis may be in the process of development. Plain radiographs of the chest may show areas of collapse or increased markings in the lung fields. If the presence of collapse can be verified, active measures should be undertaken to produce reaeration of the lung. In the older child this can usually be achieved by postural drainage, forcible coughing, the use of steam inhalations and planned physiotherapy designed to expand the bases of the lungs. The infant may be placed in a steam tent to loosen the secretions. If, after a few weeks of this treatment, there is no evidence of reexpansion, bronchoscopy should be recommended. This should be carried out under general anæsthesia, as bronchoscopic suction can remove secretions only from the main bronchi. After the introduction of the bronchoscope, the plane of anæsthesia should be lightened, so that the patient may cough, and by the exercise of his own "tussic squeeze" force secretions from the smaller bronchi, so that they may be aspirated by the sucker. The above treatment may be sufficient to cure the cough, and may in fact prevent bronchiectasis.

The diagnosis of bronchiectasis cannot be established with certainty without the assistance of a bronchogram. This procedure should be undertaken when the persisting symptoms suggest a reasonable suspicion that bronchiectasis is present. It is not proposed to discuss the technical details involved, but adequate filling of both lungs should be achieved to demonstrate the full extent of the lesion. In the earliest cases of bronchiectasis the radiological appearance of the bronchogram may be normal, but the first recognizable abnormality is a uniform or cylindrical dilatation of the affected bronchi, which are often "crowded" together indicating persisting collapse. In the later stages of the disease, saccular or cystic areas are seen in the affected segments.

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Treatment.

When the bronchogram has demonstrated a definite dilatation of some parts of the bronchial tree, an assessment must be made of the physical condition of the patient and of the extent and degree of involvement of the lungs. This is not done entirely on the radiological appearances. The degree of lung involvement is usually more extensive than is apparent in the radiograph, and this fact has been borne out by comparing specimens removed at operation with the pre-operative radiographs. Much information can be gained from the amount of sputum habitually produced by the patient, and also from the amount that can be sucked out of the bronchi prior to the performance of bronchography.

When established bronchiectasis has been demonstrated, it must be impressed on the parents that a long course of treatment will be necessary, and that results will depend largely on their cooperation and insistence that the child carries out his drill regularly.

Whatever the distribution of the disease, whether confined to one lobe or of more widespread distribution, the preliminary treatment should be the same. The most important feature of this routine is the daily performance of postural drainage. An effective means of carrying out this procedure was described in this journal by L. B. Elwell on June 18, 1949. Drainage is carried out during the night by raising the foot of the bed and making the child sleep head down. After the night's rest, the child is made to lean over the side of the bed and support himself by the hands placed on the floor. Judicious use of a "comic strip" placed on the floor will popularize this part of the procedure, which should be maintained for up to twenty minutes. The child is encouraged to cough, and tapping the chest with the border of the hand is said to help loosen the secretions. Postural drainage carried out in this manner will, in most cases, produce a fair amount of secretion, but there are some children who do not cough much at this stage. Later in the day, however, when the hyperventilation produced by active exercise causes a fit of coughing, the bronchial tree will be cleared. This draws attention to the second important part of the routine, namely, physiotherapy. Regular breathing exercises should form a constant part of the routine, and it is not sufficient for the child to go through the form of the exercise, but definite inspiratory and expiratory efforts must be made. This procedure frequently produces an attack of coughing and the production of a large quantity of secretion, which postural drainage failed to achieve.

The essence of all this treatment is to promote drainage from the affected areas and to remove the secretions which are formed. In some cases in which drainage is not satisfactory and especially in younger children, whose cooperation is not so easy to obtain, bronchoscopic suction will remove secretions and promote drainage, and it is frequently found that after such treatment better results will then be obtained with postural drainage.

This routine is a long and monotonous one, but it will be appreciated that if results are to be obtained it must be persevered with for long periods. In the treatment of the child with bronchiectasis there is no place for the familiar technique, "let me know if she is not doing well and I will see her again". The practitioner must be an aggressive force in the household, and by his own conviction that results can be achieved, convey his enthusiasm to the people concerned.

It has been demonstrated that under such a regime early cases of bronchiectasis are reversible; that is to say, the bronchogram will revert to a normal radiological picture, and the patients lose their symptoms. This fact is the justification for a long period of conservative treatment. Even if the case is not reversible, it can be predicted with confidence that there will be a great improvement in the patient's condition. Moreover, the disease will remain confined to the areas which were found to be affected at the original examination. This is not a "creeping" disease that spreads from one part of the lung to another, but the extent of lung involvement is usually determined early in the course of the disease, and it will be confined to those areas even if only minimal treatment is carried out. There may be progress from a cylindrical to a saccular condition in the involved area, but it is not usual for new areas to become affected.

Chemotherapy.

Temporary improvement can be achieved by the use of nebulized penicillin, but few reports of lasting improvement have been noted. Sulphonamides in the dosage of 0·5 gramme (one tablet) every eight hours for children over five years, and 0·34 gramme (two-thirds of a tablet) for children under five years have been used over long periods as a prophylaxis against respiratory infections with satisfactory results, but it should be emphasized that these are ancillary methods and that the essential part of the treatment is the drill outlined above.

Pre-Bronchiectasis.

There may be a period lasting up to one or two years during which bronchiectasis may be suspected by the presence of persistent cough or physical signs in the chest or persistent shadows in the radiograph, but the bronchogram is normal. This period is sometimes referred to as the pre-bronchiectatic state. As many of the children concerned are finally found to develop irreversible bronchiectasis, it is wise to consider that this condition is in process of development and to establish the routine treatment. Some will lose their symptoms and remain cured, but others will persist until finally the bronchogram will establish the diagnosis.

Prevention.

Prevention may be achieved by the careful supervision of the convalescence of children suffering from respiratory infections. Persistent cough must suggest the possibility of pulmonary collapse, and vigorous measures should be instituted to ensure reaeration of the lung. Adequate chemotherapy in the treatment of recurring bronchitis or pneumonia, the use of chloramphenicol in the treatment of pertussis and the use of pertussis vaccine as a prophylactic measure in infants can all be expected to lessen the incidence of the disease. The importance of climate should not be overlooked, and it does appear to be a fact that respiratory infections are not so common in the higher altitudes away from the coastal belt; so that both for convalescence and for treatment in the established case these conditions are desirable.

Surgery.

In the case which has been classed as irreversible the only prospect of cure lies in surgical removal of the affected lobe or lobes. In the majority of cases the conservative treatment outlined above will be designed to improve the patient's health and render him fit for operation. But the limitations of operative treatment must be realized. The ideal condition for surgery is that in which the disease is confined to one lobe (usually the left lower) or at most two lobes (lower and lingula), but these cases are the exception. If such a case is met with, there is nothing to be gained by years of conservative treatment; and when the patient is in a fit state to stand operation, lobectomy should be performed with a very good prospect of cure. This is usually performed at the age of four or five years, and it is found that children stand these operations very well. An additional reason for early operation in these cases is the fact that remaining bronchi grow and the remaining pulmonary tissue comes to occupy almost the same area as the original lung. The functional capacity improves, and this compensatory mechanism is a feature of the growing period.

Over 80% of cases, however, have been found to be bilateral, and these constitute the greatest problem. In the bilateral cases there is no doubt about the very great improvement that follows removal of the most affected portion of the lung, not only in the general health, but in a lessened production of sputum and freedom from pyrexial attacks. The patients are not cured and, after operation, should revert to their conservative treatment to drain the remaining affected parts. In a year or so, if there is still a considerable amount of sputum, further lobectomy should be considered. This represents the present feeling at the Royal Alexandra Hospital for Children, and in no case in which this plan has been followed has there been cause for regret. When there is extensive involvement of both sides, and no improvement has been achieved by conservative treatment, it is doubtful whether surgery has any place, but it is better to persevere with conservative treatment until improvement can be demonstrated.

Bronchiectasis Associated with Hilar Tuberculosis.

No mention has been made so far of an interesting group of cases, described by Williams in this journal on August 18, 1951, which were due to obstruction of a bronchus by the effects of the primary infection of tuberculosis. In these cases the patients may show little evidence of ill health and do not produce purulent sputum; so that they are not likely to be confused with the suppurative bronchiectasis described in this article.

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Conclusion.

There are still a number of questions to be answered in the study of this important disease. What is the foundation for the belief that in many cases the condition "dries up" in adolescence and causes few, if any, symptoms in adult life? Does bronchiectasis often arise as a primary disease in adult life, or does it usually have its origin in early childhood? What special factor or "tissue inferiority" determines that the incidence in females is usually twice as great as that in males? What effects on the expectation of life and activity of a child are produced by the removal of all lung tissue except the upper two lobes? In how many cases that are classed as reversible and cured in childhood does the patient develop symptoms again in later life? These questions can be answered only by continued research and follow-up studies. At present the responsibility that faces the profession is to recognize the disease and to ensure that children are enabled to receive the benefits of treatment and so dispel the gloomy pictures that are still found in the literature.

T. Y. NELSON,
Sydney.

British Medical Association News.

SCIENTIFIC.

A MEETING of the New South Wales Branch of the British Medical Association was held on May 29, 1952, at the Robert H. Todd Assembly Hall, British Medical Association House, 135 Macquarie Street, Sydney, DR. K. C. T. RAWLE, the President, in the chair.

Pruritus Vulvæ.

DR. S. D. MEARES read a paper entitled "Pruritus Vulvæ" (see page 61).

DR. J. W. FLYNN read a paper entitled "Pruritus Vulvæ" (see page 63).

DR. R. B. C. STEVENSON expressed his appreciation of the papers, and said that he would have liked Dr. Meares to make some reference to the common threadworm, which was a frequent cause of pruritus. Only two nights previously he (Dr. Stevenson) had been asked to examine a patient, eight months pregnant, at a public hospital. The patient had telephoned her own doctor and reported that she had noticed something coming down; her doctor had sent her to hospital. On examining her, Dr. Stevenson had found that she had a prolapse of the cervix through the vulva; he could see threadworms crawling all round the anus. The patient had had an itch for a long time, but no one had been able to do anything for it. Another point raised by both Dr. Meares and Dr. Flynn was the question of leucoplakia; there seemed to be doubt as to how many of those conditions were premalignant. Dr. Flynn had said that it was possible to get leucoplakia of the lip; Dr. Stevenson said one could also get leucoplakia of the tongue, and so far as he knew such lesions did not always become malignant. He had not seen many instances of *leucoplakia vulvæ* becoming malignant. He agreed with Dr. Meares that a biopsy of the vulva should be made; but the site of the biopsy should be very carefully selected. It was wrong to allow the impression to remain that *leucoplakia vulvæ* always became malignant. The question was rather whether malignant change, when it did occur, was due to the leucoplakia, or whether it was due to constant irritation by scratching. Dr. Stevenson had known cases in which leucoplakia was not associated with much itching.

DR. J. C. BELISARIO said that he wished to comment on one or two points. One had been brought out by Dr. Flynn; it concerned the use of the word "leucoplakia", which literally meant "white patch". What was referred to as leucoplakia at the present time was essentially hypertrophy, and leucoplakia was only another word for keratosis or hyperkeratosis occurring on mucous membranes. Consequently, a better term would be leucokeratosis. Referring to the comparison between leucoplakia and kraurosis, Dr. Belisario said that the article mentioned by Dr. Flynn was excellent. Leucoplakia was apparently a form of hypertrophy, and kraurosis a form of atrophy. Many now thought that kraurosis was a symptom, not a disease—a symptom following many kinds of damage, such as low-grade infections and inflammatory conditions—and that it did not occur *per se*. *Lichen sclerosus et atrophicans* was more frequent than people believed. There were one or two differentiating features between the two conditions. *Lichen sclerosus et atrophicans* appeared in the form of small macules, which fused to form continuous areas or plaques. It might be found also on the body, where kraurosis was not found. It was very important that a biopsy should be made. With regard to treatment, Dr. Belisario said that it had been found by a number of authorities that œstrogens applied locally gave relief in some cases. Some found that the relief could be continued by parenteral injection of œstrogens. Others had found that testosterone afforded relief. Vitamin *A* also had been found to be of value; it might be given orally or parenterally, or by local application. With regard to pruritus, X rays were believed to give relief because they relieved surface tension. A method that had been found to be very successful was the use of procaine base, 0·05 gramme per millilitre of water; the solution was injected under the area of local irritation. It produced anæsthesia lasting for fourteen days, so that the patient stopped rubbing and the condition cured itself. Procaine hydrochloride given by intravenous injection had been found useful; one dose could be given over twenty minutes, or a much larger dose over one and a half hours. One useful substance for application was "Anæsthesin", one drachm to an ounce of zinc ointment. By far the most useful substance for local application in *pruritus ani* was aluminium hydroxide cream. Its use in that way had been suggested by the observation that surgeons used it round colostomy openings, to prevent excoriation of the skin. Dr. Belisario said that he had had more success in many cases with that ointment than with anything else. In the last issue of *The Journal of Investigative Dermatology*, it had been reported by Fromer and Cormia that in three out of four cases of kraurosis the patients had been completely relieved with ACTH, in doses of 100 milligrammes for three days, 50 milligrammes for three days and 10 milligrammes for maintenance. It seemed as if that treatment might be useful.

DR. F. N. CHENHALL said that the speakers had gone far afield. He thought that the vulva was a very particular area; it reacted differently from the skin anywhere else. Dr. Chenhall intended to cross swords with Dr. Flynn on account of his remarks about leucoplakia of the lip and ulceration of the lip not being painful, whereas a carcinomatous ulcer of the vulva was nearly always painful. Dr. Chenhall said that he doubted whether one of the slides shown by Dr. Meares was really one of well-developed leucoplakia; he asked that one of the dermatologists present should say what it was. Dr. Chenhall agreed that gynæcologists should cooperate closely with the dermatologists before they carried out any surgical procedures in leucoplakia. Dr. Flynn had rightly stressed the general treatment of the patient. Diet and rest were most important. They did not encounter many of the cases under discussion. Dr. Chenhall said that he had tested Swift's idea that achlorhydria caused the condition, and that hydrochloric acid and vitamin *A* were useful. He had found that two-thirds of the patients tested in the out-patient department had hyperchlorhydria, not achlorhydria. *Kraurosis vulvæ* as they knew it was a very rare condition. It was wrapped up with endocrine dysfunction, and in most cases it would be better if œstrogens were used in treatment by local application. "Œstroform", 1 in 1000, gave good results in certain cases. Dr. Chenhall said that one grew tired of examining elderly women who had been given large doses of œstrogens, and who reported because of hæmorrhage. One had to make all investigations, such as curettage, to be sure that they were not suffering from malignant disease. The other type of patient who needed careful watching was the one with subclinical moniliasis. Negative results might be obtained on attempted culture, but it was important to persist. The results were excellent if the patients were watched for long enough. Patients with *leucoplakia vulvæ* which did not respond to any of the simple methods of treatment needed to be watched for a time. If after some years the condition was progressing, then it was certainly precancerous. Such patients should not be given X-ray therapy. The condition could quite easily go on to cancer, and by the time it had done so, the clinician was a long way behind in the race. The operation then required was very extensive. The Bassett-Way operation had not been followed up for long enough to allow the end results to be known. In such cases prophylaxis would be better than treatment. In odd cases, when it seemed that the condition was precancerous, it was better to act early and perform a simple vulvectomy. Dr. Chenhall said that he agreed with Dr. Flynn that it was not practicable to go round whipping off vulvæ indiscriminately.

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DR. IDA SAUNDERS referred to Dr. Flynn's doubt whether leucoplakia really caused itching. She said that it was Swift, of Adelaide, who had drawn attention to the association between leucoplakia and achlorhydria. Dr. Saunders mentioned one patient who had undergone an extensive operation for uterine prolapse, and whose itching still continued. She was found to have extensive leucoplakia, and a test meal examination revealed complete achlorhydria. The administration of dilute hydrochloric acid stopped the itching, but the leucoplakia continued to advance. Dr. Belisario had referred to the administration of vitamins *A* and *D*. At the time of Swift's work they gave crude cod liver oil. Dr. Saunders said that she went even further than Dr. Chenhall; she thought it was dangerous to stress the innocence of a condition like leucoplakia. A great deal was known about itching, compared with the little that was known about cancer. In time (up to thirty years), leucoplakia would generally become malignant; 47% of cases was the lowest proportion. All patients with leucoplakia should undergo vulvectomy. Dr. Saunders thought that leucoplakia was very uncommon; it seemed that the condition in question was often neurodermatitis. She also thought that treatment with cortisone and ACTH, if it was not kept up for more than fourteen days, was the answer to the question of treatment in these cases. However, there were underlying causes that the psychiatrists could speak about, rather than resort to Selye's shock syndrome and bring in the adrenals.

DR. J. V. MUTTON said that he had learnt a lot about leucoplakia, but although he had seen many vulvæ, he had not found many cases of leucoplakia; if the condition was there, he must have missed it. He thought it must be very uncommon. He wanted to ask a question about Epidermophyton—did that mean tinea? From his own experience of many cases of *pruritus ani* and *pruritus vulvæ*, he often found that examination of the patients' toes disclosed tinea. What was the benefit to be derived from stopping the patient from scratching? If that could be done, half the distance towards cure had been covered. He had not heard anyone mention the use of antihistamine creams.

Dr. Meares, in reply, said that one thing that had struck him about the discussion was that it would have been much easier if they had all spoken the same language. The gynæcologists did not use the same descriptive terms as the dermatologists, and *vice versa*. The only way to cure the defect was to get together. Dr. Meares hoped that he would in future be able to work in closer relationship with dermatologists. He said apparently he had not made it sufficiently clear that a vulva should be excised only when an itch had been present for a long time, when the lesion was extensive, when it was intractable, when the presence of leucoplakia had been proved by biopsy, and when ulceration and fissuring were present. He had at times removed small local patches of leucoplakia from the vulva. Willis thought that procedure very wrong, and believed that a complete simple vulvectomy should be performed. Dr. Meares had never regretted excising a vulva, and no patient had ever regretted that he had done it. If œstrogens were used in the treatment of *pruritus vulvæ*, they were better used as local applications or suppositories rather than by mouth or by injection. Dr. Meares said that Dr. Belisario had taught him a lot of dermatology, as he had done in the past. Dr. Meares was grateful for the hints about testosterone and vitamin *A*. Dilute hydrochloric acid was sometimes of great use; but achlorhydria was found in surprisingly few cases. He looked forward to trying aluminium hydroxide paste and procaine base, and possibly cortisone. Agreeing with Dr. Saunders's remarks about advanced leucoplakia, Dr. Meares said that no matter what was done, it would progress. It could progress in two ways; in 30% of cases it would go on to become epithelioma, and in the rest to cure. The difficulty was to decide in a given case in which direction the progress would be. He had not used antihistamine cream in the absence of signs of allergy. The reason he had talked so little about the treatment of the more common and less dramatic types of itch was because he knew less about them.

Dr. Flynn, in reply, said that he was glad that Dr. Stevenson had mentioned threadworms. They were very important and a very simple cause of itching. Hookworm did not often cause *pruritus vulvæ* or *pruritus ani*. Strongyloides, on the contrary, did do so. In reply to Dr. Chenhall, Dr. Flynn said that often *pruritus ani* went with *pruritus vulvæ*; it would be too much specializing to attempt cure of one but not of the other. Further in reply to Dr. Chenhall, Dr. Flynn said that he never did advocate X-ray treatment for leucoplakia; X-ray therapy was used to relieve pruritus. He deprecated its use for leucoplakia of the vulva. It was useful for neurodermatitis, *lichen planus et cetera*, many of which conditions Dr. Chenhall apparently regarded as leucoplakia. Dr. Flynn agreed with Dr. Mutton that stopping of the rubbing was the most important thing. Whatever was found suitable in the majority of cases should be used. The only proviso was to use the mildest medication possible. Dr. Flynn said that he did not find the antihistamines much use for itching, except in the treatment of insect bites. It was even possible to get insect bites in the area under discussion. Dr. Flynn thanked Dr. Saunders for her interesting part in the discussion.

Dr. Macdonald, from the chair, thanked Dr. Meares and Dr. Flynn for their papers. He said that he had found them very interesting and intensely stimulating. If those present left the meeting with their interest in this distressing condition stimulated, some of the unfortunate sufferers therefrom would benefit, and the speakers would feel repaid for their trouble and industry.

Medical Societies.

THE MEDICAL SCIENCES CLUB OF SOUTH AUSTRALIA.

A MEETING of the Medical Sciences Club of South Australia was held in the Anatomy Theatre, New Medical School, Frome Road, Adelaide, on November 7, 1952.

The Immune State of Aborigines.

DR. J. A. R. MILES gave a brief report on a trip to investigate the immune state of aborigines in the Northern Territory, which consisted largely of an outline of the methods used for obtaining representative samples of aborigines' bloods in order to test them for incidence of poliomyelitis. The remainder of Dr. Miles's talk consisted of a travelogue illustrated with "Kodachrome" slides.

Insulin Hypoglycæmia in Ruminants.

MR. I. G. JARRETT, in a paper on insulin hypoglycæmia in ruminants, said that adult sheep and young lambs showed a difference in their response to insulin when it was administered by the intravenous route. Lambs exhibited hypoglycæmic convulsions after a dose of eight units of insulin per kilogram, whereas that dose produced no obvious signs of hypoglycæmic distress in the adult. If splanchnicotomy was performed in the sheep, convulsions occurred after that dose of insulin. However, when insulin was given by subcutaneous injection, the effect in the adult sheep was more dramatic than in the lamb and did not support the contention that sheep were insulin resistant. A dose of four units per kilogram would usually cause convulsions in a young lamb about five or six hours after the injection, and in an adult sheep about twelve or fifteen hours after the injection. However, whereas the lamb always recovered, the sheep, once convulsions had started, was usually in an irreversible state, and several injections of glucose often failed to relieve the condition.

Butyric Acid and Insulin Hypoglycæmia.

MR. B. J. POTTER, in a paper on butyric acid and insulin hypoglycæmia, said that the lower fatty acids, acetic, propionic and butyric, had been widely studied in relation to intermediary metabolism. Propionic acid was known to be glucogenic, but acetic and butyric acids had usually been considered as intermediates in fat metabolism. The intravenous administration of the sodium salts of these acids to adult sheep indicated that acetate caused no alteration in the total amount of reducing sugar in blood compared with a pronounced increase following administration of butyrate or propionate. Circulating ketone bodies were increased in amount after administration of acetate or butyrate but not propionate.

Hypoglycæmic convulsions induced in lambs with insulin were promptly relieved by an intravenous injection of either sodium butyrate or sodium propionate with a concurrent increase in the blood sugar content. Acetate neither relieved the convulsions nor caused any elevation in blood sugar level.

Mr. Potter said that the effect of butyrate did not appear to be the result of a sympathetico-adrenal discharge, because convulsions were still relieved in lambs after bilateral splanchnicotomy to denervate the adrenals. Butyrate and

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propionate were also effective in relieving convulsions in adult ewes in which the splanchnic nerves had been sectioned, but acetate was again ineffective. Investigations on non-ruminants indicated that butyrate caused cessation of convulsions in adult rabbits with a corresponding increase in blood sugar content. It was suggested that butyric acid might, under certain conditions, be associated with carbohydrate metabolism.

MELBOURNE PÆDIATRIC SOCIETY.

THE monthly meeting of the Melbourne Pædiatric Society was held at the Children's Hospital, Melbourne, on Wednesday, August 13, 1952.

Idiopathic Myocarditis in Infancy and Childhood.

DR. HOWARD WILLIAMS read a paper on idiopathic myocarditis in infancy and childhood and presented the clinical records of 13 patients. He pointed out that comparatively little was known about the clinical features of the disorder as the majority of cases that had been described had been rapidly fatal, and that it was usually the pathologist and not the clinician who established the diagnosis. Eight of the 13 patients under discussion had presented with acute heart failure, which proved fatal in seven. The remaining five patients developed cardiac failure more slowly, and four of those recovered.

Dr. Williams said that the acute cases presented the following typical clinical, radiological, electrocardiographic and pathological features. The initial manifestations of the malady were commonly mild. Either one or a combination of a group of sypmtoms (malaise, drowsiness, refusal to feed, vomiting, loose stools, pallor) was usually present for several days or a few weeks. During that time the patient was usually afebrile, but occasionally the temperature was elevated 1° or 2° F. The symptoms were usually attributed by the mother to a minor ailment, but the development of rapid, grunting, laboured breathing and cyanosis compelled her to seek medical aid urgently.

Examination disclosed a seriously ill child, with severe circulatory and respiratory embarrassment, whose restlessness and apprehensive look suggested pneumonia with anoxæmia. In addition to the cyanosis, pallor was often quite pronounced, and the fingers, lips and ears often had a greyish appearance. The respiratory rate was rapid, frequently as high as 90 to 100 per minute, and a short expiratory grunt or slight wheeze was often heard. The chest was rather barrel-shaped, the percussion note was resonant, and at times the upper margin of liver dulness was displaced downwards one or two intercostal spaces. The breath sounds were harsh, and in some patients crepitations were heard at the lung bases. The extremities and face were usually cold, the skin was mottled, and the capillary circulation was very sluggish. The pulse was thready, the rate was rapid, usually between 150 and 170 per minute, and the blood pressure was low. Cardiac enlargement was always present, but often could not be detected clinically, as the rapid respiratory rate and restlessness of the infant made palpation of the apex beat impossible, and the emphysematous chest rendered percussion of the heart unreliable. Sometimes enlargement was suggested by palpable cardiac pulsation immediately below the xiphisternum in the epigastrium. The heart sounds were usually of poor quality, the second sound at the apex often being louder than the first, and in some gallop rhythm further indicated cardiac embarrassment. Venous engorgement in the neck could not be determined with any degree of certainty in many patients because of restlessness and the short stumpy neck present in most infants. The liver edge was invariably three or four fingers' breadth below the costal margin, and could be overlooked unless palpation was commenced well down in the iliac fossa below the liver edge. The spleen was palpable in a few patients. Subcutaneous œdema was very variable; in some it was not present, in a few only the sacrum and legs were œdematous, while two patients had œdema of the face and hands as well. In a few patients the urine contained a trace of albumin. The overall clinical picture was that of an anoxæmic infant with severe circulatory failure and considerable respiratory distress. To the uninitiated the picture might suggest severe pneumonia with cyanosis and circulatory failure.

Radiological examination of the chest showed a gross uniform cardiac enlargement. In contrast to the large heart the lung fields were usually relatively normal, and in only a few cases was there any appreciable venous engorgement. In several, small bilateral pleural effusions were noted, and in two an area of radio-opacity at one or other lung base suggested collapse, consolidation or both. The electrocardiogram showed serious impairment of myocardial function, significant features being low voltage *QRS* complexes and low isoelectric or inverted *T* waves.

Post-mortem examination showed that all hearts were dilated, and usually the walls were thicker than normal. The only other macroscopic abnormality noted was some flabbiness of the heart muscle with irregular reddish or pale streaking in the muscle wall. With one exception the pericardium, endocardium and valves were all normal. Histological examination revealed widespread but frequently patchy damage of the myocardium, and associated inflammatory reaction with neutrophile and eosinophile polymorphonuclear leucocytes, lymphocytes and macrophages.

The subacute type of lesion presented a similar pattern, but the onset was more gradual, and four or five patients seen survived after a long illness; however, all four had permanent cardiac enlargement. While it was not possible to prove that the four patients who survived had an identical pathological lesion, the clinical, radiological and electrocardiographic features were the same.

In conclusion, Dr. Howard Williams said that myocarditis of unknown ætiology was a serious, frequently fatal malady, which usually occurred in the first two years of life, but could affect infants a few weeks of age and also older children. The pathological lesion consisted of widespread but usually patchy muscle damage with an associated cellular inflammatory reaction. The common clinical pattern was an abrupt onset of fatal cardiac failure, which was manifested by severe respiratory embarrassment, cyanosis, tachycardia and an enlarged liver and heart. The illness in some cases was more insidious in onset and the heart failure not so severe; recovery could occur, but usually with permanent heart damage. The acute form of the disorder was commonly mistaken for pneumonia, as respiratory embarrassment and cyanosis were the outstanding clinical features, and cardiac enlargement was difficult and frequently impossible to detect clinically. Treatment with digitalis and oxygen was of definite value in some patients. The clinical and pathological evidence did not reveal the cause, and the ætiology was unknown.

DR. A. L. WILLIAMS discussed the pathological aspects of myocarditis in childhood. He said that acute myocarditis of unknown ætiology had been responsible for the deaths of at least nine children in the Children's Hospital during the past three years. During that period 635 autopsies had been performed. The nine cases in which death occurred appeared to be examples of isolated myocarditis, for there was no evidence that the occurrence of the lesion was secondary to any other disease process. In three of the children an associated respiratory tract infection was observed.

At autopsy evidence of congestive cardiac failure was always present. In the majority of cases the heart was enlarged, owing to dilatation of the ventricular cavities. The myocardium was characteristically flabby, often with a mottled pallor. Pericardial fluid was usually present in excess, but no macroscopic lesions of valves or endocardium were encountered. A definite diagnosis could not be established until sections of the myocardium were examined, when histological examination revealed patchy but diffuse areas of myofibril necrosis, with an associated inflammatory response. The areas where fragmentation of muscle fibres had occurred appeared œdematous, and were infiltrated by leucocytes. In most sections examined lymphocytes and neutrophile and eosinophile polymorphonuclear leucocytes were the predominant cells, but occasionally plasma cells and macrophages were present in quantity.

DR. M. L. POWELL opened the discussion. He said that the condition of idiopathic myocarditis was being diagnosed more frequently lately, but had undoubtedly occurred in the past without being recognized in many cases. Dr. Bowden had demonstrated that sudden death in infancy from unknown causes, often thought to be the result of suffocation, was frequently the result of myocarditis.

Dr. Powell mentioned the rare occurrence of cardiac murmurs in the condition under discussion compared with the frequency of gallop rhythm. That was probably due to the myocardial weakness with inadequate muscular contraction.

The use of digitalis was recommended in all cases, but dramatic results should not be expected. As in many cases the cardiac rate was not increased, the value of digitalis in slowing the rate was not prominent.

The extreme damage to the myocardium seen in the subjects who came to post-mortem examination made it obvious that a long convalescence was usually necessary.

DR. KATE CAMPBELL quoted two recent outbreaks in newborn infants in maternity hospitals. In one outbreak, five babies had recovered, but two had died, and post-mortem examination performed by Dr. K. M. Bowden confirmed the diagnosis of myocarditis. More recently two newborn infants had developed the condition which clinically simulated *asphyxia neonatorum*. One infant had died, but the other recovered. An interesting feature was the fact that both mothers suffered from "colds" which had been present during the weeks immediately preceding and also at the time of delivery. Dr. Campbell said that she suspected an association between the mother's infection and the infant's myocarditis and the possibility of intrauterine transmission of infection.

DR. ROBERT SOUTHBY said that he agreed with the suggestion of virus infection. Outbreaks seemed to occur in waves, supporting the theory of an epidemic nature of the disease.

DR. KEITH BOWDEN said that the disease was much commoner both in children and in adults than was generally realized. In 230 cases of sudden death in children under the age of six years who had been taken to the City Morgue, acute myocarditis had ranked fourth in frequency, being outnumbered only by cases of respiratory tract infection, meningitis and meningococcal septicæmia. That served to demonstrate the importance of the disease, but practitioners were not aware of its frequency, and the diagnosis was rarely made before death. The condition could rarely be diagnosed on macroscopic examination of the heart, microscopic sections usually being necessary.

DR. H. MCLORINAN said that acute idiopathic myocarditis was probably the result of an acute infection, the causative organism of which was most likely to be a virus. However, the clinical features in many ways resembled those of acute infections due to *Hæmophilus influenzæ*, such as acute epiglottitis, in which the obstruction was not the cause of death. In a number of the cases microscopic evidence of myocarditis was present and the lungs were normal. Dr. Ferris had been obtaining positive cultures of *Hæmophilus influenzæ* from the blood of every patient clinically diagnosed as suffering from the condition. Dr. McLorinan suggested that blood cultures carried out on patients diagnosed as suffering from myocarditis might produce a growth of *Hæmophilus influenzæ* in a percentage of cases.

Dr. Alan Williams said that he agreed that blood culture should be carried out. However, a large percentage of the results were negative, probably owing in many cases to the widespread use of antibiotics. In experimental animals, Coxsackie and allied viruses caused myocardial lesions similar to those demonstrated in the cases under discussion.

Dr. Howard Williams said that antibiotics were not indicated unless there was reasonable evidence of bacterial infection. Dr. Williams asked if there was an explanation for the absence of pulmonary congestion.

DR. H. B. KAY, in replying to Dr. Howard Williams, said that the explanation was not completely known, but was probably mechanical in nature and related to the chest and heart sizes. In children, the chest size was relatively less than the heart size. However, the same problem presented in adults—left ventricular failure presenting as right ventricular failure, with an absence of pulmonary congestion. Dr. Kay said that in the previous twelve months he had met with 12 cases of myocarditis in adults. Many were in the younger age group—under thirty years. Diarrhœa was a common presenting symptom. There was a group which developed branch bundle block. Those patients usually developed congestive cardiac failure after a period of twenty to thirty years. Many patients, forty to fifty years of age, who developed congestive cardiac failure and were diagnosed as suffering from ischæmic heart disease with branch bundle block, were probably suffering from the sequelæ of myocarditis originating in childhood. Referring to the treatment of myocarditis, Dr. Kay said that a combination of digitalis and quinidine was probably more effective than either given alone.

Persistent Vomiting and Failure to Thrive with a Mechanical Basis.

DR. MURRAY CLARKE presented two patients who had been suffering from recurrent persistent vomiting and failure to thrive.

The first patient was a boy, aged two years, the youngest of three boys, his brothers being sixteen and ten years old. The father had had a nervous breakdown, and his mother had undergone thyroidectomy twelve months before the child's birth, whilst one month after his birth she had developed an attack of blindness due to "nerves". The patient's birth weight was nine pounds. Vomiting three or four days in the week had persisted from birth until the time of coming under observation with only two remissions, the longest being three months. Discomfort but not pain accompanied the vomiting. The vomitus was always green, the vomiting being usually projectile or in a large gush. Vomiting occurred two or three hours after meals and often at night. On one occasion sultanas taken twenty-four hours previously had been seen in the vomitus. The patient's appetite was good, but at fifteen months of age he refused milk, and it was said on his admission to hospital that he always vomited milk, cheese and eggs. He weighed 24 pounds at the age of twelve months and 28 pounds at the age of two years.

Dr. Clarke said that six weeks before the patient's admission to hospital vomiting had become more persistent, loss of weight was noticed, and there was slight abdominal distension. A barium meal examination showed only dilated stomach with retention of barium after four hours. Laparotomy disclosed a distended stomach and duodenum, a volvulus of the small intestine and the presence of a mesentery for the large intestine. The volvulus was undone and the space between the jejunum and colon widened to prevent a recurrence of the volvulus.

The second patient was a girl, aged five years, who when examined weighed only 27 pounds. She had failed to thrive from birth, gaining only three ounces in the first six weeks of life. Feedings were taken well, but most of the food was returned one hour later and was bile stained. Recently vomiting had become more persistent and occurred usually once in twenty-four hours at night. The longest time for which food was noted to be retained was three days. The vomiting was painless. Laparotomy revealed an abnormal disposition of the gut and a greatly dilated stomach and duodenum twisted on itself, with a septum at the duodenojejunal junction causing obstruction. This was excised and a local anastomosis performed. The child gained five pounds in three weeks after operation.

Dr. Murray Clarke pointed out that in both the patients there had been a mechanical cause for which one might anticipate a favourable surgical correction. He then outlined the normal embryological development and the abnormalities which had resulted in the two children having an extrinsic and an intrinsic obstruction respectively. He said that the majority of such intestinal obstructions were acute and could be diagnosed in the first few weeks of life; the minority were usually diagnosed later, and very often the patient had abdominal pain of acute onset which often subsided equally rapidly. In a few cases, the two reported being examples, the patient had vomiting without pain. The clue to diagnosis was that one or all of the symptoms—pain, vomiting, failure to thrive—were atypical and not characteristic of any of the well-known syndromes. If a mechanical disorder was borne in mind and investigated it might be possible to salvage some from the "rubbish heaps" of indigestion, cyclical vomiting and chronic appendicitis. Perhaps more careful histories, more frequent radiological examinations and more exploratory laparotomies would be necessary to achieve that happy result.

DR. ELIZABETH TURNER opened the discussion and pointed out the difficulties in diagnosis of the first patient presented by Dr. Murray Clarke. Dr. Turner said that she had examined the child previously. There was a strong neurotic background, which confused the diagnosis, and the mother's original history differed from that which she subsequently gave. The vomiting at night did not support a nervous origin. Later, clinical features including abdominal distension favoured the diagnosis of mechanical obstruction. Dr. Turner then outlined the conditions with which mechanical obstruction could be confused and stressed the importance of an accurate history.

DR. F. E. PLARRE demonstrated the radiological features of the second patient shown by Dr. Clarke. He said that she was a mongol, aged five years, and was smaller and more stunted than her sister, aged two years. In addition to the long history of vomiting, strong visible gastric peristalsis had been observed in the out-patient department by the examining physician. The patient was a very cooperative and bright mongol. After her stomach had filled there was a considerable delay in the opening of the pylorus, despite posturing, and a small beak-like projection was seen, suggesting either pyloric stenosis or pylorospasm. After fifteen minutes the barium passed on freely to fill

another sac in the right upper quadrant, apparently a very dilated, atonic and obstructed duodenum. The pylorus opened with free communication backwards and forwards between stomach and duodenum, but with an elongated channel of variable calibre, some films showing twisted folds. The pyloric obstruction was therefore assumed to be due to extrinsic bands or a volvulus rather than intrinsic stenosis.

The barium was held up in the dilated duodenal bulb for over an hour and then slowly and steadily trickled through to the jejunum and small bowel. This was all situated on the right side, and the caecum was over to the left of the mid-line, the whole colon being entirely left-sided.

Dr. Plarre said that the presence of malrotation with abnormal mesenteric attachments having been established, it was assumed that the duodenal obstruction was also extrinsic. In retrospect, however, it was obvious that the appearance of the ballooned duodenum was characteristic of an intrinsic pin-hole diaphragm, and in view of Bodian's finding of the frequent association of mongolism with cases of neonatal duodenal obstruction due to abnormal septa, the presence of a double lesion of both intrinsic stenosis and malrotation might have been anticipated.

DR. ROBERT SOUTHBY said that in his experience many cases of so-called "cyclical vomiting" turned out to be cases of intestinal obstruction, and he rather doubted whether there was such an entity as cyclical vomiting.

Dr. Turner, in reply, said that she considered that fat intolerance was a basis for vomiting occurring in cyclical episodes, and therefore represented cyclical vomiting of a *non*-obstructive origin.

Out of the Past.

In this column will be published from time to time extracts, taken from medical journals, newspapers, official and historical records, diaries and so on, dealing with events connected with the early medical history of Australia.

QUARANTINE AGAINST SMALLPOX.[1]

Colonial Secretary's Office,
Sydney,
4 August, 1828.

ROBERT DUNN, Esq.,
Surgeon Supt.,
Ship BUSSORAH MERCHANT.

Sir,

I am directed by His Excellency the Governor to inform you that the Troops and Prisoners on board the BUSSORAH MERCHANT are to be landed with the necessary utensils and the Berths and Bulkheads on a point of land in Spring Cove[2] and lodged in tents until all danger of infection be removed. A small vessel called the Alligator will be stationed at the nearest convenient place, and the whole are to be considered as Still in a state of Quarantine.

You will be pleased therefore to continue in the medical charge, but to place yourself under the orders of Captain Daveney, who as the Officer Commanding the Detachment will be instructed to see the Quarantine Regulations enforced.

A cordon of sentries will be posted so as to prevent all intercourse by land and the Master of the Dart will be instructed to intercept all communication by water and all letters will of course be required to be fumigated as before—should any vinegar or other article be required for this purpose you will be pleased to transmit a Report to that effect.

I have, &c.,
ALEXR. MCLEAY.

Colonial Secretary's Office,
11 September, 1828.

Robt. Dunn, Esq.,
Spring Cove.

Sir,

The medical officers having reported that there is no longer any disease among the People at Spring Cove, but that it will be proper to guard against infection being conveyed by the Bedding or Clothing, I am directed by His Excellency the Governor to inform you that the whole are to be released from Quarantine, brought to Sydney by Boats, and the Prisoners distributed as assigned on Saturday next. But Captain Daveney has received Instructions to cause the following precautionary measures to be carried into effect, viz.:

The Clothing of the Soldiers is to be carefully cleansed and such articles as circumstances will permit are to be thoroughly soaked in water.

The Clothing used by the Convicts while on board is to be *wholly destroyed*, as well as that of the child which had the smallpox, and of its parents. Care is also to be taken that the new Clothing issued to the Convicts is not allowed to come in contact with the old articles.

The *Whole* of the *Bedding* both of the Soldiers and Convicts is to be *destroyed*.

You will be pleased therefore to give every assistance that may be necessary in carrying these orders into effect.

I have, &c.,
ALEXR. MCLEAY.

[1] From the original in the Mitchell Library, Sydney.

[2] The site of the present Quarantine Station on the North Head of Port Jackson.

Correspondence.

A NEW EXCURSION INTO ETHER ANÆSTHESIA.

SIR: I think that most anæsthetists will disagree with several of the suggestions made by Dr. R. B. Lynch in the journal of December 6, 1952, particularly in regard to patient safety. The following points are of immediate interest and importance:

1. The method of vaporizing ether by blowing a stream of gas over or through the liquid is as old as the storage of gases in containers, and in particular was used by Boyle as a standard procedure early in this century.

2. The use of a high concentration of oxygen in an anæsthetic mixture, whilst allowable, is dangerous, in that it can only too readily mask the onset of respiratory obstruction with its associated increased respiratory effort and retained carbon dioxide. For this reason it should be eschewed by the occasional anæsthetist.

3. The practice of heating ether by standing the bowl in hot water is to be condemned. Boiling ether has caused deaths from acute pulmonary œdema when it has subsequently condensed in delivery tubes or the lungs themselves.

4. The suggestion that "Carbogen" should replace oxygen as a vaporizing vehicle must also be condemned. Dr. Lynch states that his patients look very well when they go back to bed. Presumably they have hypertension and peripheral vasodilatation, which go hand in hand with hypercapnia. One wonders if they look so well an hour or two post-operatively when they have blown off the excess carbon dioxide and are hypotensive and pale. Furthermore, the excessive oozing which accompanies the peripheral vasodilatation must be annoying to the surgeon, and no less disturbing to the patient.

Yours, etc.,
IAN H. MCDONALD, M.B., B.S., D.A.

Children's Hospital,
Carlton,
Victoria.
December 9, 1952.

SIR: The search for simplicity in anæsthetic apparatus is laudable, provided that known safety factors are not ignored. Herewith some criticism of the apparatus described (THE MEDICAL JOURNAL OF AUSTRALIA, December 6, 1952) and some fallacies indicated.

1. No provision is made for trapping liquid ether between the vaporizer and the point of delivery to the patient. The obvious requirement is a trap-bottle.

2. The amount of heat conveyed into the vaporizer by passing a stream of oxygen through a coil immersed in hot water is negligible and will aid vaporization but little. The only safe method of increasing the vaporizing capacity of an ether vaporizer is by immersion in a water bath (at a temperature below the boiling point of ether) or, in a more cumbersome way, as in the Oxford vaporizer.

3. One deprecates the use of oxygen as a substitute for adequate lung ventilation in the management of cyanosis during anæsthesia. The respiratory system is attuned to air with an oxygen concentration of 21%. The addition of ether vapour to air lowers the oxygen concentration. The addition of oxygen to restore the 21% is advisable. Except in a limited number of pathological conditions, 21% oxygen is adequate. When cyanosis accompanies the inhalation of a 21% oxygen atmosphere, the fault is usually not the oxygen concentration but the tidal respiratory volume, and the obvious treatment is the restoration of normal tidal respiration, if necessary by assistance to respiration—a provision not supplied in this apparatus. When assisting respiration, it is wise to ensure that the increased volume of air is passing into the lungs and not into the stomach. The treatment of cyanosis during anæsthesia by the giving of high concentrations of oxygen is based on a fallacy. The cyanosis is only one side of the picture—the visible side. By using high concentrations of oxygen, the oxygen needed by the patient is readily available, and the cyanosis may disappear with a tidal volume totally inadequate for the removal of carbon dioxide, leading to retention of carbon dioxide. Ventilation with air in sufficient volume to provide the required oxygen ensures clearance of carbon dioxide.

4. A number of spontaneous ether explosions have been reported overseas in which static electricity was the igniting agent. In some, at least, of these accidents, the anæsthetic used was oxygen blown over ether. In this apparatus, dry oxygen at a high pressure passes through a reducing valve causing friction, which is a potent factor in the generation of static electricity, then "warmed" in the coil, thence to the ether container where it picks up ether vapour, giving a highly explosive mixture. One hopes that the new excursion into ether anæsthesia does not lead to a further excursion—an explosive one. The hazard of "oxygen over ether" may and should be eliminated by vaporizing the ether with air and adding oxygen to the air-ether mixture. The dry oxygen in this method does not pass through the ether container.

Yours, etc.,

DOUGLAS G. RENTON, F.F.A.R.A.C.S.

Melbourne,
December 7, 1952.

SIR: We wish to draw your attention to the anomaly which exists in regard to Dr. R. B. Lynch's article on page 808 of your issue of December 6, 1952. It seems strange that such an article should appear at length in this year 1952, when the papers presented to the Section of Anæsthesia at the recent Australasian Medical Congress (British Medical Association) appeared only in abstract.

We respect Dr. Lynch for his enthusiasm and for his obvious efforts to improve the standard of anæsthetics in a rural locality. At the same time, we would point out in a friendly spirit that: (a) The basis of his apparatus is really Shipway's, adopted for field service in 1915 and abandoned by even the Australian army before the second World War. (b) Modification of Shipway's design by warming the air before passing it through the ether has been tried long ago, sometimes with disastrous results. (c) The use of "Carbogen" (which did not, we trust, contain 10% of carbon dioxide!) was abandoned as unphysiological as long ago as 1938.

We could add other criticisms, but enough has been said to show that it would be a pity if the article in question were to be taken as in any way representing current ideas upon anæsthetics in Australia in 1952.

Yours, etc.,

N. R. JAMES.
R. H. ORTON.
G. KAYE.

49 Mathoura Road,
Melbourne,
December 10, 1952.

SIR: There is nothing new in the method of giving ether described by Dr. R. B. Lynch in your issue of December 6, 1952. The idea of using compressed oxygen or "Carbogen" for the double purpose of providing a vehicle for ether vapour as well as the motive power for its insufflation has been current for many years. The chief thing that is wrong in attempting to combine ether with oxygen in this way is the risk of a violent explosion, which would almost certainly be lethal to the patient, and perhaps to bystanders as well.

Static electricity generated by the passage of the gaseous mixture through rubber and other tubes could cause ignition, despite the absence of other sources of sparking. The risk might perhaps be lessened if the oxygen were first passed through a humidifier, but Dr. Lynch's apparatus makes no provision for this. Even so, air is a far safer vehicle than oxygen for the inflammable anæsthetic agents and if required, extra oxygen should be added to the mixture somewhere close to its point of delivery.

Nowadays compressed air is as readily available as oxygen, and would be preferable for Dr. Lynch's purposes. Failing this, a dental foot-bellows will provide a powerful stream of air for insufflational procedures. Alternatively, recourse may be had to "inhalational" devices like the Oxford vaporizer and the Flagg "can". Nothing could be simpler or cheaper than the latter, which together with a suitable Magill tube will provide satisfactory anæsthesia for dental and oral surgery in most adolescent and adult subjects. This also permits the use of a secure pharyngeal pack, so necessary in the prevention of serious lung complications after such work.

Dr. Lynch's apparatus cannot possibly satisfy the ideals which he outlines in his paper. His method is certainly not applicable to all types of case, nor is it better and safer than many others. At least he does warn of the danger of overheating the ether and of the possibility of its condensation in the delivery tube, but the absence of a trap-bottle in this line is incredible. Further, to restrict free breathing in order to increase or maintain the depth of anæsthesia is surely a physiological and technical outrage.

Yours, etc.,

S. V. MARSHALL.

143 Macquarie Street,
Sydney,
December 13, 1952.

SIR: In Dr. Lynch's article "A New Excursion into Ether Anæsthesia" (December 6, 1952) there are a number of statements on which comment could well be made.

He states rightly that safety is the prime consideration. This ether apparatus is unsafe, in that the ether jar could be easily upset resulting in neat ether being delivered to the patient. There is no ether trap bottle, nor is there a safety valve to blow off in the event of an excessive rise in pressure in the system.

Dr. Lynch suggests using 100% oxygen for the insufflation. Although this may keep the patient a good colour, it increases the fire risk from that of burning to that of detonation.

Most anæsthetists agree that "Carbogen" has little place in the modern techniques. The overbreathing induced may cause (i) rise in blood pressure, (ii) sweating, (iii) tiring of the respiratory muscles, and (iv) upset of acid-base balance.

The article states that the apparatus is used for dental extractions. No mention has been made of a throat pack, so there is the possibility of inhaling a foreign body. The patient should be intubated, packed off and an Ayre's "T" piece, or similar arrangement, used to connect the tube to the apparatus.

Dr. Lynch says: "In all but the oral or transoral procedures keep the mouth shut. . . ." This could result in obstructed breathing. The patient cannot fill his lungs from three litres of oxygen a minute and must breathe round the nasal catheters. This would be especially marked with large catheters.

To say that "this system is eminently applicable for all surgical anæsthesia" is unwise. How could it possibly be used with an open chest or many other operations which one could enumerate?

Surely an "Oxford vaporizer" is reasonably cheap, safe, reliable and simple to use?

Yours, etc.,

WINSTON SMITH.

143 Macquarie Street,
Sydney,
December 29, 1952.

CONSULTANT SERVICE FOR LEGACY CHILDREN.

SIR: The Medical Committee of Sydney Legacy would like to bring to the notice of medical practitioners that many families of deceased ex-servicemen are ineligible for repatriation medical benefits. Many such families are

enrolled in Sydney Legacy, but may incur medical expenses because of the illnesses of their children, and to some this may represent a considerable financial burden.

The Committee therefore wishes to bring to the attention of practitioners the free consultant service for Legacy children available through Sydney Legacy, to children resident in Sydney, and to country children through the appropriate country Legacy organization via Sydney Legacy. Consultants, both members of Legacy and otherwise, will see Legacy wards in their rooms, or at their clinics at public hospitals, and investigations can be carried out at the latter for no charge. Should an operation be necessary, the Medical Committee of Sydney Legacy can arrange for this to be done at a public hospital so that no professional fees are involved.

Arrangements for this consultant service may be made through the Almoner, Sydney Legacy (MA 9155), or through the Chairman, Medical Committee.

It is hoped that this letter will stimulate practitioners to cooperate in this matter, and so reduce financial obligations by Legacy widows on behalf of their children, commitments which they are so often forced to hand to Legacy.

In conclusion, the Medical Committee of Sydney Legacy would like to take this opportunity of thanking those many practitioners who have cooperated with them so wholeheartedly in the past.

Yours, etc. (for Sydney Legacy),

S. J. M. GOULSTON,
Chairman, Medical Committee.

144 Castlereagh Street,
Sydney,
December 17, 1952.

THE AUSTRALIAN ABORIGINES AND OURSELVES.

SIR: May I thank you for the editorial of November 1, 1952. As one who has for over twenty years personally investigated the potentialities of the Australian aborigines—tribal natives in the heart of the continent and in the tropic north, full-bloods on cattle stations, missions and government compounds, and part aborigines in the settled country of the south—I am in a position to support your contentions.

The approach to the aborigines has too often been wrong even when well meant. Little heed has been given to mental development. "Teachers" until recently have nearly always been untrained. By contrast, the Ernabella Mission in the Musgrave Ranges, which I initiated, has trained experts in charge of every phase of the work, and in fifteen years has proved beyond doubt that aboriginal children are as educable as white children, and that the younger adults of both sexes can step into our civilization with success, given expert training and encouragement. At Ernabella the entire work of the station is now done by the people themselves with a minimum of supervision even to woolclassing. Without any semblance of compulsion the whole tribe has successfully and with happiness entered a new world while still maintaining its tribal customs. The two races at this new village in tribal country are complementary—each with full respect for the other.

The University of Sydney has the honour of creating the first scholarship for an aboriginal student, but others are on the way. The National Union of Australian Students through Melbourne and other universities has this year made investigations into the matter of such scholarships. The question of resident scholarships at university colleges must now be considered. A women's college in Melbourne has already made an offer stipulating that, if possible, two students should come at the same time. There need be no qualms about admitting aborigines to corporate life. For many years aborigines—full blood and part—have lived in my home as full members, and for seventeen years I have kept in touch with the young men and women who have come to Adelaide from the interior. My secretary is one of them. She is able, accurate and courteous and takes more responsibility off my shoulders than anyone has in forty years. She is the daughter of a tribal mother of the bush. When opportunity for aborigines widens I have no fear of the result, but let it be remembered that the aborigines are like ourselves—a few brilliant, a few dull, but the great majority in between. All, however, at times are made conscious of the attitude of superiority which many white Australians, including New Australians, adopt towards them. That attitude, which you have so courageously attacked, is keeping back even very able aborigines from their full place in the community.

In closing, I would point out that the full-blood population in Australia is now increasing where opportunity and respect are the order of the day. The supply of amino-acids in their diet is not as liberal as ours!

Yours, etc.,

CHARLES DUGUID.

Adelaide,
December 11, 1952.

ACUTE MASTITIS: A STUDY OF 28 CASES.

SIR: Dr. Isbister's article on acute lactational mastitis (THE MEDICAL JOURNAL OF AUSTRALIA, December 6, 1952) brings to prominence one aspect of the entire problem of satisfactory, or unsatisfactory, breast feeding. Her indication that infection of the baby precedes, and leads to, mastitis in the mother is of obvious significance. However, the object of this letter is to stress the other factors involved: breast engorgement and nipple injury. Harold K. Waller, of the British Hospital for Mothers and Babies, London, S.E.18, has studied the importance of these factors for many years. It was a privilege to work under Waller and to become acquainted with an institution which has a breast feeding record unsurpassed in England.

Dr. Isbister quotes three of Waller's articles. The reader is referred to these, for in matter of detail no better commentary exists than the author's own writings.

In matters of principle there are three points which need stressing:

1. Waller's management begins at the first ante-natal visit and finishes six months after the baby is born. He has a staff which is devoted to his methods (albeit after much opposition) and carries out a routine so thorough that it would possibly be beyond the capabilities of any nursing staff who consider a forty-hour week as one of the ideals of living. Waller has developed that team spirit which is essential to the success of any combined operation.

2. Apropos the matter of routine: Dr. Isbister remarks that there "can be no routine method of handling the problems of lactation". If I were to say, on the contrary, a routine is absolutely essential, we would probably both be correct in so far as there must always be small, individual variations from any routine. But to have a good routine is the very essence of the matter.

3. Dr. Isbister states: "Success depends on the ability and desire of the patient to cooperate." This is also true in so far as it goes, but there is a very marked tendency on the parts of many doctors and many nurses, when failure is imminent, to unload the responsibility on to the patient. Waller himself asked a large meeting of midwives and health visitors for the causes of failure in breast feeding. Their answers, in order of importance, began with worry and maternal unwillingness. As Waller commented, most of the reasons given can be classified as sociological, and he went on to examine the real, the physiological, causes.

I would like to add that the patient's response depends largely upon the attitude and competence of the obstetrician, the pædiatrician, the nursing staff and the welfare clinics, and upon the atmosphere they create—of harmony, of confidence in a scheme in which all believe, of thoroughness, of anticipation, and of unremitting vigilance.

The hospital at which Waller worked is known to its visitors from all over the world, not for its obstetrics (which is good), but for its handling of lactation.

Yours, etc.,

ARCHIE G. MURRAY.

7 Malcolm Street,
Perth,
December 12, 1952.

COLLES'S FRACTURE.

SIR: In the correspondence column of your issue of February 2, 1952, you published a letter from myself protesting against the term Colles fracture, asking particularly for the meaning of the term, whether it meant any particular fracture of the lower extremity of the radius *et cetera*. These questions have not so far been answered, but in a recent article by Dr. A. R. Hamilton in the issue of this

journal for December 13, 1952, the protest is completely ignored, although in his introductory paragraph he seems to show that Colles's fracture is not the fracture described by Colles in these words: ". . . whereas Colles considered the fracture to occur approximately one and a half inches from the distal end of the radius, later observations and X-ray examination have shown that the fracture is usually about one inch from the articular surface. Furthermore, Colles described only the simple fracture and omitted reference to the comminuted fracture, which is of frequent occurrence."

In view of the fact that most practitioners include all fractures in this neighbourhood as Colles's fracture and that apparently great diversity of opinion exists amongst surgeons, orthopædists, radiologists and others as to what actually constitutes such fracture, would it not be far more intelligible, accurate, less confusing and more in accordance with modern scientific knowledge to abandon this term and substitute the far more easily understood descriptions, such as fracture of the lower extremity of the radius, with or without involvement of the wrist or inferior radio-ulnar joint, with a description of such deformities as may be present, whether "dinner fork" or other and any other of the very many complications which may be noted in the individual case? Unless this is done, the use of the term will continue to confuse everybody, even the laymen and patient himself.

It is hoped that senior specialists and others may be induced to drop the term, and until this is done, it is unlikely that juniors and students will follow suit, for the variety of fractures in this neighbourhood is very great and can be diagnosed only by radiographic measures and understood by descriptions in ordinary intelligible language.

Yours, etc.,

H. FLECKER.

52 Abbott Street,
Cairns,
Queensland.
December 20, 1952.

ORANGE POST-GRADUATE INSTRUCTION COURSE, MARCH 9-13, 1953.

SIR: There seems to be some confusion over the date of this course and I would like to take this opportunity of stating it is the week Monday to Friday, March 9 to 13, 1953.

We hope to publish a full programme later on, but it is based on the following: a demonstration of clinical cases at the Orange Base Hospital, a session at the Orange Mental Hospital, a session on diseases of animal origin and some fifteen to twenty lectures. The specialist lecturers who have so far cooperated are Professor B. T. Mayes (Department of Obstetrics, University of Sydney), Dr. K. B. Noad (honorary physician, Sydney Hospital) and Dr. G. R. A. Syme (honorary surgeon to in-patients, Royal Melbourne Hospital). There are several organized tours and the official dinner.

Yours, etc.,

S. R. DAWES,
Honorary Organizing Secretary.

Orange,
New South Wales,
January 7, 1953.

Obituary.

JOHN DALE.

WE are indebted to Dr. Frank Scholes for the following appreciation of the late Dr. John Dale.

John Dale was born on May 2, 1885, at Coleshill, a small market town eight miles from Birmingham. He was educated at Solihull Grammar School near by and at the University of Birmingham, graduating M.B., B.S. in 1908 and taking the M.R.C.S., L.R.C.P. in the same year. He proceeded to the B.Sc. (Public Health), Birmingham, in 1909. From his university he gained a Walter Myers Travelling Studentship, and during its tenure from 1909 to 1911 he visited Germany and other Continental countries. Returning to Birmingham, he was appointed assistant medical officer of health, which position he retained till 1919. He spoke often of his work in and about Smethwick, a rather depressing industrial suburb. Just as his earlier travels fostered his love of music and the arts, so it is probable that his work in these poor districts helped to bring about the constantly humane attitude that distinguished his later activities in larger fields.

John Dale served through the first World War as major, R.A.M.C., from 1914 to 1919. He was D.A.D.M.S. (Sanitation) to the Second and Fourth Armies, and the Army of the Rhine, and for his services was awarded the O.B.E. in 1918. Once again he returned to Birmingham and took his M.D. degree in 1919.

He came to Western Australia late in 1919 and held the position of medical officer of health in that State until 1927, when he was appointed medical officer of health to the City of Melbourne, in succession to the late T. W. Sinclair.

John brought a fresh mind and outlook, not only to his council and department and to those of us concerned with public health and hospital work, but to the general public. He was not particularly interested in the formal, legislative or conventional aspects of his office, but he was enormously interested in people, in how they were fed, clothed and housed, and in all ways of keeping them healthy. His earliest efforts were directed towards provision of health services for mothers and children, and in his first year of office he had succeeded in creating the position of child welfare medical officer, to which Dr. Hilda Kincaid was appointed. Before his term expired the original small services had been augmented by the provision of four kindergartens and eight additional baby health centres, so distributed throughout the city that no mother would need to travel more than a quarter of a mile to reach a centre. Within a year also Dr. Hilda Bull had been appointed to deal with problems associated with infectious disease in the city, including immunization against the dangerous diseases of childhood. Later a dental officer was appointed to work at pre-school centres.

John worked hard in the cause of reclamation of slums in the city and was greatly disappointed that so little came of his recommendations. He showed a lively and practical interest in all aspects of slum life, not only in the city proper, but throughout the metropolitan area. In

fact that lively interest showed itself in innumerable ways, and he wrote, lectured and broadcast on an immense variety of topics connected with public health. He was the joy of newspaper reporters, for not only was he friendly and accessible, but invariably they went away with the germ of an arresting and sometimes provocative "par". As President of the Victorian Branch of the Health Association of Australia he wrote a great number of pamphlets under such titles as "A Food Dialogue", "The Septic Focus" and "Coughs, Colds and Children". He never indulged in newspaper controversies, but set the ball rolling for many.

One of his greatest interests was the provision of an adequate diet and proper nutrition, both from a physiological point of view and, as the depression years 1929-1933 advanced, as a matter of economics. He tabulated diets and costs, which were widely read and studied, and prescribed a standard extraction rate for "wholemeal" bread. The "Dr. Dale loaf" became well known and obtainable in many shops throughout the country.

John Dale was a member of practically every important committee in the State which was connected in any way with public health.

From 1929 to 1950 he was Lecturer in Public Health at the University of Melbourne. From 1928 to 1950 he was a member of the Council of the Victorian Branch of the British Medical Association, and in 1945 was President of the Branch. At one time he was Chairman of the Section of Preventive Medicine.

He was a member of the Food Standards Committee, a Chairman of the National Fitness Council, on the Committee of the Victorian Board of Examiners for the Royal Sanitary Institute, and Council representative of the Victorian Baby Health Centres Association.

On occasions of difficulty, governments have found it necessary to appoint consultative councils with considerable responsibility and advisory powers. During 1937-1938 John was a member of the State Consultative Council on Poliomyelitis, and in 1943 of that on typhoid fever. He was a pleasant and helpful colleague, sane and moderate in discussion.

He lived a very full life and his was a familiar figure in many non-medical settings. He was an omnivorous lover of the arts in all forms, from modern abstract art to pottery. He looked in at most exhibitions of paintings, etchings and handicrafts and officially opened quite a number. The theatre was one of his chief joys, particularly opera, French, Russian, German and Italian. He often dropped in to lunch-hour organ recitals in the Town Hall and rarely missed an important orchestral concert or recital of chamber music. Not often was his lank figure seen in evening dress; almost invariably he wore a not very new suit of an indescribable brown colour. He liked a quiet meal in Bohemian surroundings, or to listen to a few gramophone recordings in a private house. His favourite outdoor sport was trout fishing.

In appearance John was very long, lanky and angular, with shoulders somewhat stooped. When he rose from the depths of an armchair to greet his hostess, or to reach for his raincoat to go home, it seemed that a most complicated set of levers was put into operation. Someone described it as the appearance of a jack-knife unfolding. His features were good, especially in profile, with fine brows, and his smile made him handsome. He was an excellent singer of German *Lieder* and had a beautiful speaking voice, which added to the charm of his conversation, but he was not forceful enough to be an orator, and his failure to enter parliametary life was no tragedy, for he would not have been at home there.

John relinquished his post as medical officer of health for the City of Melbourne in 1950. Shortly after his marriage with Dr. Hilda Bull, who had been for so long associated with him in the City Health Department, he accepted a Commonwealth post in Holland, his duty being the supervision of examination of prospective Dutch migrants. He and his wife, whose environment throughout her life had been literary and artistic, and who shared his tastes, enjoyed a leisurely journey through Italy *en route* for Holland. In the months that followed they both paid short visits to Britain and in the autumn of 1952 went on a holiday trip to Italy. On September 27 John was killed when the car in which they were travelling overturned near Venice.

He will be remembered with affection by hosts of friends and by many men and women who never even met him. His public work in Melbourne will be remembered, and it still goes on, particularly in the domain of health and happiness of women and children.

Dr. Arthur Brown writes: It is one of the pleasant memories of my life that I knew John Dale well enough to call him my friend. Many people knew him by sight, and no one who saw this tall, lean, dark man, with his strong rugged face, his long rebellious hair, and heard his deep resonant musical voice, could help feeling that in him there dwelt a character out of the ordinary run. And very true that was. His voice was one of the most beautiful speaking voices I have ever heard, and its deep music, either when he was declaiming in unmeasured language against some social injustice, or even more when he was talking over some plan for assisting the campaigns against disease or the alleviation of the effects of poverty, enhanced the feeling of sincerity and the conviction of his sympathy which were evident in the words he used.

Essentially John was an artist in the truest sense. I once introduced to him a friend from New Zealand, a highly cultured surgeon. After we left him, my friend said to me almost in wonderment: "Why, he's a poet." He was a poet, who never wrote poetry. An artist who never painted. But he was at home in the atmosphere of poetry and painting and in the friendship of artists, as few of us are who are outside the magic circle. He had, too, the artist's intolerance of the ordinary rules which bind and limit us, and cramp our actions. He had the classicist's love for forlorn hopes and lost causes. He made a gallant, lone, unendorsed and unsupported attempt on a seat in the Senate; and he found the same attraction in the financial theories of Major Douglas as did A. R. Orage and other sensitive men in England. He declaimed against controls, against almost all controls, having an unusually deep belief and trust in the integrity of the individual. Any suggestion that he, or we, or anyone was being, in his favourite phrase, "pushed around" would rouse him to fury, a fury that expressed itself in very forcible phrases, but ended soon in his kindly smile. Those of us who sat in Council with him know of the electric atmosphere that was apparent as soon as he stood up to speak. One never knew what he would say next, but one always knew that it would be said with a force and a humour which few of us could emulate.

In his own chosen line of work he was tireless and efficient, always seeking the underlying cause of the things he disapproved, never being satisfied with attacking symptoms only. For that reason he will not be remembered for positive achievements, in spite of his campaign for the healthier loaf with which his name was for a time associated, but for the deep thought and the work which he devoted to eradicating the causes of social as well as bodily diseases. I well remember the deep earnestness with which he would say to me when talking of the handicaps of our underprivileged children: "But have you *seen* them? Have you *been* through Fitzroy and Collingwood yourself? You'll never forget them if you once go there and *see* them." Great individualist as he was, he was never individualist in his work, which always held the picture of the underprivileged as a class before his eyes.

He was an unusually fine character, and of a picturesqueness of the sort that enriches the society in which it dwells. One cannot help feeling that the world is poorer for his untimely end.

WILLIAM EWART GYE.

We are indebted to Dr. J. L. Day for the following appreciation of the late Professor William Ewart Gye.

William Ewart Gye, F.R.S., B.Sc. (London), M.D. (Edin.), F.R.C.P., Director of the Cancer Research Laboratory of the Department of Public Health, Western Australia, died in Perth on October 14, 1952. He was born in Derbyshire in 1884. Circumstances forced him to leave school at the age of fourteen years, and during the next few years he was engaged in many and varied occupations. While a pupil teacher he came under the influence of a gifted and enthusiastic schoolmaster, who inspired him with an intense appreciation of literature and the classics. He passed the London matriculation examination in 1902, and entered the University of Nottingham to study for his London B.Sc. Always a keen cricketer, he became a professional for a time in order to supplement his meagre income. He graduated B.Sc. (London) in 1905, winning a scholarship at the University of Nottingham. Here he worked on the synthesis of the silicon compounds, from which later were developed the silicones, now of great importance in industry.

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He was next appointed lecturer in chemistry, at the Aston Technical School, where he worked with Frankland on the process of oxidation of metals. Finally the ultimate goal was reached, and he qualified in medicine at Edinburgh in 1912, winning innumerable prizes and scholarships on the way. He remained in the pathology department at Edinburgh, perfecting his research technique, until 1913, when he joined the staff of the Imperial Cancer Research Fund, under the directorship of first the founder, Dr. Bashford, and later his successor, Dr. Murray.

Through the first World War, he served with the Royal Army Medical Corps in France. Here his researches continued, sometimes actually in the trenches, as when he investigated tetanus and demonstrated the need of calcium chloride in the soil for infection. He was mentioned in dispatches. In 1919 on his return from the war he was appointed to the staff of the Medical Research Council, where he continued to work on general pathological problems. Among many other subjects, he investigated gas gangrene and canine distemper, and made the fundamental discoveries in the elucidation of the pathology of silicosis. At this time he began to work on the virus origin of cancer, starting from the Peyton Rous discovery of the filtrable tumours of chickens.

In 1935, on the retirement of Dr. Murray, Gye was invited to accept the directorship of the Imperial Cancer Research Fund, then located in old laboratories in Queen's Square. His first move was the designing and building of the present laboratory buildings at Mill Hill, which for functional efficiency and beauty of design will long remain a memorial to him. Work there was in full swing at the outbreak of the second World War. He immediately turned over part of the laboratory to the Ministry of Supply for essential war researches, but continued his work on cancer at full pressure at the same time. In spite of air raids, the laboratory continued to function, and he consolidated his work on the virus origin of mammalian tumours. By the end of the war he was able to demonstrate the transmissibility of mammalian tumours by long frozen and by dried tumour material, and to point out the extreme importance of the use of cold in the investigation of the "continuing cause" of cancer. Unfortunately he reached the retiring age in 1949, with a vast programme of experiments still uncompleted. For reasons of health he retired to Western Australia, where he organized and directed a cancer research laboratory for the Public Health Department in Perth. This functioned both as a research centre for the training of staff and the establishment of breeding of special strains of animals, and also as a testing station for new methods of treatment. For this he collaborated with the staff of the Royal Perth Hospital. The interest aroused by his work has since led to the organization of an institute of cancer and general pathological research for Western Australia. It has also led to the establishment of other colonies of experimental animals in the Commonwealth, and has stimulated active public support for an aspect of medical work not previously widely known. His wife, Professor Ida Mann, was on his staff and will continue to work in the laboratory and for the firm establishment of the institute, which was unfortunately only at its inception at the time of his death.

Gye's original papers include a monograph on cancer and more than seventy papers on chemistry and pathology, with special reference to lipoid chemistry, silicosis, canine distemper, disseminated sclerosis, tetanus, and viruses and cancer in general.

Many prizes and honours came his way, including the Medal of the Royal College of Surgeons of England, the Gold Medal in Edinburgh for his M.D. thesis, and the Theobald Smith Medal in America. He was widely read in English, French and German, and he lectured in the United States on two occasions.

William Gye was an original thinker, passionately devoted to the search for scientific truth. Much of his career was concerned with the problem of cancer, and he accepted the challenge to the end. Essentially modest and retiring, and shunning publicity, he was not widely known amongst the profession in Western Australia. Those of us who were privileged to know him mourn the loss of a distinguished scientist and scholar and a great man.

LAURA MARGARET HOPE.

WE are indebted to Dr. Helen Mayo for the following appreciation of the late Dr. Laura Margaret Hope.

Dr. Laura Hope (*née* Fowler) died on September 14, 1952, at the age of eighty-three years.

In 1886 Laura Margaret Fowler passed her senior public examination, first class, in preparation for entrance to the University of Adelaide, then in its beginning. One of the provisions of the new university had met with some opposition from the Colonial Office, namely, that men and women should be admitted on equal terms. However, this was passed in 1881, leaving the way legally clear for Miss Fowler to enter the medical course. She was an excellent pioneer in this quest, being intelligent and indeed gifted, attractive, with wavy brown hair and blue eyes, and a quiet and dignified manner. She had an earnest desire and determination to do good work.

Her university course was successfully achieved without rebuff and she graduated M.B., B.S. in 1891, the first of her sex in South Australia to obtain this degree. Then she became a resident medical officer at the Adelaide Children's Hospital, the board being timorously courageous about the new departure of appointing a woman doctor, and a year later inordinately proud of the success of their experiment. After this, she went to Sydney to do a course in midwifery, and on July 4, 1893, she married Dr. Charles Standish Hope. He, having taken his B.A. in Cambridge, had come back to Adelaide and graduated M.B., B.S., in 1889, taking his M.D. among the first four to gain this degree in Adelaide in 1891.

After their marriage they sailed for India, arriving at Calcutta on July 31 and proceeding forthwith to Pabna in North Bengal as medical missionaries. Here, there was already a Baptist mission centre, and these doctors began at once to organize the medical side of the work. Here they remained for the next forty years, except for leave periods, for the taking of a course in tropical medicine at Liverpool in 1902, and for the 1914-1918 war, when they joined the Scottish Women's Hospital. The accompanying photograph of Dr. Laura in the uniform of the Scottish Women's Hospital (uniform grey with Gordon tartan trimmings) was taken in 1915. Dr. Laura was in charge of a hospital unit in Serbia, Dr. Charles being the only man in the organization. They were taken prisoner with the Serbian army and suffered many hardships in their return across Europe.

In Pabna, the medical work was most efficiently organized. Every day at 6.30 a.m. patients were interviewed. The most urgent 40 patients were given tickets and were seen that day; those who were less serious were given tickets for the next day. A full clinical examination was made of each patient including blood tests *et cetera*. At 10.30 a.m. there was an interval for breakfast, after which the work was resumed. After the midday siesta there were many preparations to be made for the next day—compounding of drugs, sterilization of dressings *et cetera*. In the cool of the evening visits were paid to sick patients.

During the first years, the patients came from all classes of the population, but as Indian medical training became more efficient the more wealthy patients went to their compatriots. There were, however, always many poor sick people who came to the missionaries, who were known and trusted throughout the whole district. The diseases treated were the tropical complaints, malaria, kala azar, the dysenteries *et cetera*, as well as ordinary medical and surgical conditions, including eye diseases, such as cataract, and complaints of other climes, including the exanthemata.

Dr. Laura was the better linguist, while Dr. Charles excelled in surgery and mechanics generally. The excellence of their work was recognized not only by the people—the Government of India awarded Dr. Charles the silver Kaisar-i-Hind Medal after twenty years' service and Dr. Laura the gold Kaisar-i-Hind Medal in 1933.

Dr. Laura kept diaries and wrote accounts of many aspects of the work in India. She also made exquisite little sketches in indian ink of houses, huts and workers in various trades. The women of Pabna presented her with an address when she received her gold medal. I quote from the concluding sentences:

> What garland—what gift can we offer to lay at your feet?
> We find no way of showing our gratitude to the one whose love and kindness have captivated us.
> For one who is the incarnation of kindness and compassion.
> For one who brings peace to those consumed in the flames of affliction a garland is not meet—garlands fade away.
> Store for her within your hearts, nectar.
> We bow before you in gratitude.
> The people of Pabna have found rest in the shadow of your love.
> We shall never be able to forget you.

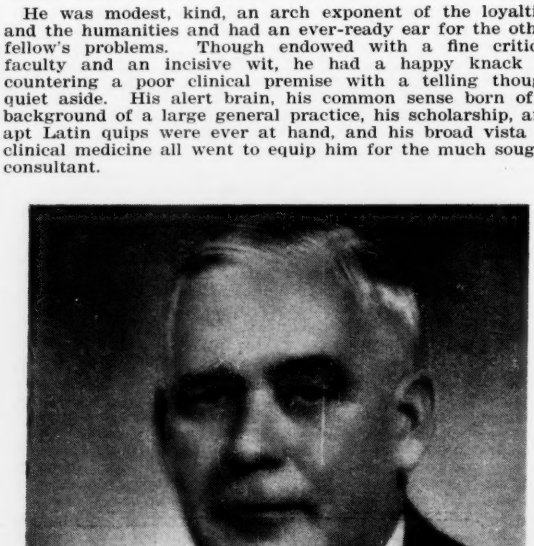
ROBERT JOSEPH TAYLOR.

DR. ROBERT JOSEPH TAYLOR, whose death was announced in a recent issue of the journal, was well known and beloved as a consulting physician in Sydney. His early schooling was undertaken at Sydney Grammar School and he studied medicine at the University of Sydney. He distinguished himself at school in modern languages, obtaining the Lithgow Prize at the senior public examination. At sports he was a champion miler. As an undergraduate in medicine he took a prominent part in university life. He acted as representative of his year throughout his course and he was elected president of the Undergraduates Association, an honour which was highly prized. He qualified as Bachelor of Medicine in 1914 and became resident medical officer at the Royal Prince Alfred Hospital. Here he became associated with the late Robert Gordon Craig. Taylor joined the Australian Army Medical Corps and served with the Australian Imperial Force from 1915 to 1919. He was mentioned in dispatches in 1918. When Robert Gordon Craig was appointed commanding officer of the hospital ship *Karoola*, Taylor joined his staff. He left the *Karoola* and became regimental medical officer to the 4th Battalion, which was then commanded by Lieutenant-Colonel (now Lieutenant-General, Sir) Iven Mackay. He won the respect and affection of those with whom he was associated and later was attached to the 9th Australian Field Ambulance. He returned to Australia after the first World War and commenced practice at Randwick. He subsequently practised as a physician in Macquarie Street, Sydney, and became honorary physician at Saint Vincent's Hospital and also at the Royal Alexandra Hospital for Children. He was a foundation Fellow of The Royal Australasian College of Physicians.

Those who knew him well describe him as a loyal friend and a man full of humour and kindliness, who was always seeking for ways and means of helping other people. The sympathy of many friends and patients has been extended to his wife and his two sons.

Dr. W. J. McCristal writes: It would be safe to say that no one has been more universally held in professional esteem and affection than R. J. (Bobbie) Taylor, and equally correct to say that no one crowded more into his daily routine.

He was modest, kind, an arch exponent of the loyalties and the humanities and had an ever-ready ear for the other fellow's problems. Though endowed with a fine critical faculty and an incisive wit, he had a happy knack of countering a poor clinical premise with a telling though quiet aside. His alert brain, his common sense born of a background of a large general practice, his scholarship, and apt Latin quips were ever at hand, and his broad vista of clinical medicine all went to equip him for the much sought consultant.



A particular devotion for many years was the City Mutual Life Assurance Society, Limited, founded in the last century mainly by his relatives, J. P. Garvan and George Crowley, and of which he was a director and chief medical officer. His many and essential attributes found full scope in this field, to which he brought a high standard of performance.

"Bobbie's" ever hurried and bustling gait was proverbial and a source of chagrin to many who wanted to hear and see more of him. Yet this was inevitable since he was honorary physician to Saint Vincent's Hospital and the Royal Alexandra Hospital for Children in between his consultant specialist and life assurance and repatriation work.

The varied assemblage at his requiem at the Basilica was indicative of the wide range of his interests and the aura of his exuberant spirit prompted one to muse, as he would, in contemplation of some fine intellect ploughed up like any other brain by the vascular episode that killed him.

To his wife and his two sons, his brothers and sisters, our deep sympathy is extended.

HENRY ARTHUR SUNDSTRUP.

THE following appreciation of the late Dr. Henry Arthur Sundstrup has been sent by Dr. L. Halberstater.

Dr. Henry Arthur Sundstrup, who died in Sydney on November 22, 1952, after a protracted illness, was educated at the Gympie High School. He obtained the first open scholarship to the University of Queensland by a high school product, and graduated B.Sc. in 1917. In 1919 he obtained his M.Sc. degree. He went on to a medical course at the

University of Sydney, graduating M.B., Ch.M. in 1924. He then did his residency at the Brisbane General Hospital, and proceeded to Wondai, Queensland, which was then beginning to be developed as a farming area. Here he built up a reputation for attention to duty, honesty of purpose, and general medical and surgical ability. In 1932 he bought a large general practice in Townsville, where he practised until the outbreak of war. He then joined the Royal Australian Air Force, with which he served until the cessation of hostilities, when he returned to Townsville. Chronic ill health forced him to retire from practice in 1949. Despite the nature of his affliction, and knowing full well the seriousness of his condition, he maintained a spirit of hopefulness. He is survived by his wife and five children, who are living in Sydney.

To those who knew him intimately Arthur was a personality. His attention to his patients and devotion to duty were the key words of his life and the reason for his success. He had a wide knowledge of ancient and modern history, and was an expert on current affairs. His quick wit made him an excellent companion. His patients always referred to him as "Sunny", reflecting the bright happy outlook he had on life. I knew him as a student, as a resident, as an honorary medical officer, and as a fellow practitioner for many years. His character and work were such that he endeared himself to all. He will be sadly missed.

ELIZABETH KENNY.

SISTER ELIZABETH KENNY, whose death occurred at Toowoomba, Queensland, on November 30, 1952, was known far and wide for her activities in the field of poliomyelitis. At our request Dr. Jean Macnamara has written the following tribute to Miss Kenny's life and work.

Progress in medicine and advancement in the care of patients suffering from disease or disability result from the efforts of persons of two types: (*a*) those who, over long periods, patiently observe and record and quietly seek the basic knowledge necessary for the understanding of the principles and the basis for the details of treatment or care; (*b*) those who, like crusaders, battle for the application of knowledge to the individual patient. The time lag between the contribution of the first group of workers in poliomyelitis and the widespread application of care based upon their principles has been inordinately long. In many parts of the world the work of Charles Fayeth Taylor in 1867 is still unknown. In others, the recognition of lesser degrees of paralysis before the development of deformity is comparatively new. The quiet work of the Kendalls of Baltimore, of Jessie Stevenson in Chicago, of Colin MacKenzie in Australia interested few people. Even in centres where their basic principles have been accepted, there has been wide variation in the details of management of patients. There has never been any "orthodox treatment".

It is premature to assess whether Elizabeth Kenny has made any original contribution to the basic knowledge necessary for the care of people paralysed by poliomyelitis. Workers in different centres take different views depending on the depth of their experience before seeing Miss Kenny's work. On the other hand, her contribution as a crusader has been outstanding. The publicity which accompanied her movements and her statements has in twenty years brought the Cinderellas—the "Polios"—to be front page news.

F. D. Roosevelt commenced the blitz on the apathy which in many centres had resulted in needless crippling for paralysed people. In Australia every person paralysed by poliomyelitis and members of the teams who work to salvage them have reason to thank Elizabeth Kenny for the improved facilities available in 1952. For example, during the five epidemics which occurred between 1925 and 1931, the after care of paralysed people was exceedingly difficult because of finance. Hospitals for adults resented the hold-up of their beds for their care. The Children's Hospital (Melbourne) had many other commitments. During the depression years, one physiotherapist to supervise the care of forty children was all we could afford. Money for bare essentials such as "prams" and even boots was hard to find. The publicity which Elizabeth Kenny aroused has been a major factor in altering this state of affairs. Most governments realize that an epidemic of poliomyelitis calls for the prompt and generous expenditure of public money if crippling is to be prevented or reduced. More people in authority appreciate the economy of spending money to restore youth to efficiency by early care as opposed to payment of pensions for decades to persons unnecessarily crippled. Miss Kenny's stormy career, the spectacular claims reported in the early days in the lay Press, her book, the radio and the film aroused the public interest. The controversy between the relative merits of her method and what she termed "orthodox treatment" helped many people to realize that the old idea that an attack of poliomyelitis necessarily meant permanent crippling was false. The public in Victoria today appreciate the variation in degree of severity of the paralysis at the onset, and they know that, for the majority, given prompt, detailed, and continued care, the outlook for recovery is good. Elizabeth Kenny's contribution was more than the effect of her publicity on the loosening of purse strings. She provided a stimulus to research workers to abandon the servile adherence to the theory of essential neurotropism of the virus and the olfactory nerve-olfactory tract-axonal spread which made the thirties so sterile of progress in knowledge of human poliomyelitis.

Miss Kenny endeavoured to anticipate deformity and reduce fatigue of weakened muscles by insisting on bed rest for a long period when muscles of the trunk and lower limbs were involved. She tried to hold the trunk and limbs in comfortable positions compatible with ultimate good posture. She used many contraptions which, though rarely of metal in her early days, could fairly have been called splints. In each centre where she worked a staff was employed large enough to police the positions of the patients and their limbs.

Miss Kenny did great service to paralysed people by drawing attention to the harm which follows the practice of confining patients in splints in institutions which, through staff difficulties, could not arrange for movements, active or passive, to be carried out at intervals short enough to ensure at first attainment, then maintenance of the normal range of joint movement—a difficulty which does not arise when the patient is cared for at home by a parent coached by a physiotherapist.

Miss Kenny, with ample justification, stressed the harm which could be done by perfunctory or careless splinting, by the use of ill-designed or ill-fitting appliances. To every honest worker her comments have reverberated as a stimulus to more evaluation of the timing to reduce the degree of splinting, more detailed criticism of alignment. To some of us she has indirectly acted as a spur to bring to the manufacture of splints recent advances in technology improving appliances.

In all these aspects Elizabeth Kenny's forceful personality has resulted in far-reaching benefit. When the period of care needed for any disability is long, the majority of people tend to lose the enthusiasm and interest in detail evinced by every doctor in the care of patients needing more spectacular care. For over fifteen years Miss Kenny's attitude has been to destroy complacency.

It is interesting to speculate whether Miss Kenny's contribution would have been greater or less if early in her career her gifts of patience, pertinacity and attention to detail had been coopted by those of us working in Australia for the same objectives. The precedent had been given by the recognition of similar qualities in Agnes Hunt by Sir Robert Jones. Their work together improved orthopædic care throughout Great Britain. The offer was made to Miss Kenny at a conference in Canberra in April, 1936, but, by that time, the Press had cast her in the role of an "outsider" and Miss Kenny made her decision to retain it. It is possible that the role she selected has resulted in benefit to more paralysed people than she could have helped quietly caring for a number of patients in Australia. Fame, travel, Press interviews, her book, the film controversy were her choice, and perhaps suited her temperament better. Great benefit would follow the advent of a similar crusader for the present Cinderellas among crippled people—those with dystrophy and amyotonia, and the people suffering from multiple sclerosis: a crusader to arouse interest in their problems as Elizabeth Kenny has done for people paralysed by poliomyelitis.

Notice.

THE Section of Neurology, Psychiatry and Neurosurgery of the New South Wales Branch of the British Medical Association will hold a meeting in the Robert H. Todd Assembly Hall, British Medical Association House, 135

Macquarie Street, Sydney, on Thursday, January 29, 1953, at 8 o'clock p.m. Dr. Stuart Morson will read a paper on some observations on recent advances in neurology and neurosurgery overseas. All members of the Association are invited to be present.

Post-Graduate Work.

THE ROYAL INSTITUTE OF PUBLIC HEALTH AND HYGIENE.

THE Royal Institute of Public Health and Hygiene conducts recognized courses of instruction (starting annually in March and October) for the examinations of the Conjoint Board of the Royal College of Physicians of London and the Royal College of Surgeons of England for the certificate in public health; courses will commence on March 20 and October 2, 1953. These lead to courses (commencing in January and August) for the diploma in public health, and similarly (in February and July) for the diploma in industrial health. Students are also prepared for the diploma in industrial health examinations of the Society of Apothecaries of London.

Any of the courses may be taken whole time or part time. They consist of both lectures and visits.

Candidates desiring a course for either the diploma in public health or the diploma in industrial health of the Conjoint Board are required by them to produce evidence of being in possession of a recognized certificate in public health.

For the certificate in public health, lectures are given by specialists on the various sociological aspects, and in the course for the diploma in public health, the practical work is carried out at a county borough. The diploma in industrial health course entails visits to all types of factories and to coal mines *et cetera*.

Further information, entry forms and prospectuses may be obtained from the Secretary of the Institute, 28 Portland Place, London, W.1, or from the Dean, at 23 Queen Square, London, W.C.1.

THE MELBOURNE PERMANENT POST-GRADUATE COMMITTEE.

PROGRAMME FOR FEBRUARY.

Course for Candidates for Higher Degrees.

A COURSE in anatomy suitable for candidates for Part I M.S., primary F.R.A.C.S. and F.R.C.S., Part I D.A., D.O., D.L.O., D.P.M., D.D.R. and D.T.R.E., will commence on February 2 at 2 p.m., and will continue on Mondays and Wednesdays until August. Classes will be conducted in the Anatomy Department, University of Melbourne. The fee for this course is £15 15s., payable to the Post-Graduate Committee.

Courses in physiology, pathology *et cetera* for candidates for higher degrees, diplomas and fellowships will commence in March, and details will be published later.

A course in hæmatology suitable for candidates for M.D. Part II and M.R.A.C.P. will be conducted at different hospitals at 2 p.m. on Tuesdays and Fridays, under the direction of Dr. John Bolton. The fee for this course is £3 3s., payable to the Post-Graduate Committee.

Country Demonstration at Ballarat.

On February 26 at 8 p.m., at Craig's Hotel, Ballarat, Dr. I. H. Cuming will lecture on "Pre-Operative and Post-Operative Management". The fee for this lecture, 10s. 6d., is payable to Dr. J. P. L. Griffiths, 22 Errard Street, North, Ballarat.

Demonstration at Flinders Naval Depot.

On Wednesday, February 11, at 2.30 p.m., at Flinders Naval Depot, Dr. B. P. McLoskey will lecture on "Poliomyelitis: A Social Problem", by arrangement with the Royal Australian Navy.

Lectures by Sir Allen Daley.

Sir Allen Daley, sometime Chief Medical Officer of the County of London, who is visiting Australia under the auspices of the Nuffield Foundation, will lecture at the Warrnambool Hospital on the evening of Saturday, February 14, and at Ballarat on the evening of Tuesday, February 17. Local arrangements are in the hands of the subdivisional secretaries.

Medical Prizes.

PRIZE FOR MEDICO-SURGICAL FILM.

THE annual prize offered by *La presse médicale* for a medico-surgical film, amounting to 100 francs with various other awards, will be presented during the last session of the course of *"Actualités medico-chirurgicales"* at the *Faculté de médecine de Paris* on March 24, 1953. The prize is awarded only to amateurs for films not previously published, not subsidized and not produced by any laboratory or firm. Films will be judged on their instructional as well as their cinematographic qualities. They may be of the silent or sound variety, in colour or in black and white, but they must be of 16-millimetre size. The last day for reception of films is March 10, 1953. Applications should be sent as soon as possible and be addressed to the Secretary, *La presse médicale*, 120 Boulevard Saint-Germain, Paris.

Naval, Military and Air Force.

APPOINTMENTS.

THE undermentioned appointments, changes *et cetera* have been promulgated in the *Commonwealth of Australia Gazette*, Number 81, of December 11, 1952.

AUSTRALIAN MILITARY FORCES.

Regular Army Special Reserve.

Royal Australian Army Medical Corps.

The following officers relinquish the provisional rank of Captain and are transferred to the Reserve of Officers (Royal Australian Army Medical Corps (Medical)) (1st Military District) in the honorary rank of Captain, 22nd September, 1952: QX700169 J. H. Joyce and QX700170 F. H. Garlick.

The following officers relinquish the provisional rank of Captain and are transferred to the Reserve of Officers (Royal Australian Army Medical Corps (Medical)) (2nd Military District) in the honorary rank of Captain: NX700393 R. H. Keatinge, 9th September, 1952, NX700395 F. M. Stackpool, 16th September, 1952, and NX700373 J. W. Skalla, 27th September, 1952.

Citizen Military Forces.

Northern Command: First Military District.

Royal Australian Army Medical Corps (Medical).—The following officers are appointed from the Reserve of Officers, 10th April, 1952: 1/61806 Lieutenant-Colonel M. R. Gold, with regimental seniority next after 1/39015 Lieutenant-Colonel E. P. Row, and with pay and allowances of Major at own request, and Captains 1/61809 J. F. Gillogley and 1/61808 R. H. Orr. To be Major and Honorary Lieutenant-Colonel, 23rd September, 1952: 1/59168 Robert Kenneth Wilson. To be Captain (provisionally), 4th August, 1952: 1/61807 Robert Calder Sandford Jackson.

Eastern Command: Second Military District.

Royal Australian Army Medical Corps (Medical).—2/127808 Captain (provisionally) B. L. V. McLaughlin relinquishes the provisional rank of Captain and is transferred to the Reserve of Officers (Royal Australian Army Medical Corps (Medical)) (2nd Military District), in the honorary rank of Captain, 21st August, 1952. To be Captain (provisionally) and Temporary Major, 1st October, 1952: 2/165473 John Ernest Dunlop Goldie. To be Colonel, 1st July, 1951: 2/146504 Lieutenant-Colonel G. N. Young, E.D. (in lieu of the notification respecting this officer which

appeared in Executive Minute No. 183 of 1951, promulgated in *Commonwealth Gazette* No. 78 of 1951).

Southern Command: Third Military District.

Royal Australian Army Medical Corps (Medical).—The provisional appointment of 3/101010 Captain S. H. Watson is terminated, 31st October, 1951. 3/101010 Captain (provisionally) S. H. Watson is seconded whilst undergoing post-graduate studies in the United Kingdom, 1st November, 1951. 3/101010 Captain (provisionally) S. H. Watson ceases to be seconded for post-graduate studies in the United Kingdom, 9th September, 1952. 3/101021 Honorary Captain J. S. Penington is appointed from the Reserve of Officers, and to be Captain (provisionally), 9th September, 1952. To be Captain (provisionally), 1st November, 1951: 3/101010 Stanley Henry Watson, with regimental seniority next after 3/73277 Captain (provisionally) J. W. Bennett.

Central Command: Fourth Military District.

Royal Australian Army Medical Corps (Medical).—4/31994 Captain (provisionally) T. D. Manthorpe relinquishes the provisional rank of Captain and is transferred to the Reserve of Officers (Royal Australian Army Medical Corps (Medical)) (4th Military District) in the honorary rank of Captain, 29th September, 1952.

Reserve Citizen Military Forces.

Royal Australian Army Medical Corps.

2nd Military District.—Honorary Captains E. H. Freidman, E. P. Armstrong and P. V. Dixon are retired, 15th October, 1952.

3rd Military District.—The resignation of Captain I. D. Wilson of his commission is accepted, 7th October, 1952.

4th Military District.—Captain J. A. R. Miles is granted the honorary rank of Lieutenant-Colonel, 23rd July, 1952. To be Honorary Captain, 14th October, 1952: Robert Britten-Jones.

6th Military District: To be Honorary Captain, 8th October, 1952: Keith Samuel Millingen.

Retired List.

The following officers are placed upon the Retired List (2nd Military District) with permission to retain their rank and wear the prescribed uniform, 15th October, 1952:

Royal Australian Army Medical Corps.

1st Military District.—Lieutenant (Honorary Colonel) F. G. Scoles, E.D., Lieutenant-Colonels R. G. Quinn and N. L. Sherwood, and Honorary Major W. H. Steel.

2nd Military District.—Colonel (Honorary Brigadier) W. P. McCallum, C.B.E., D.S.O., M.C., E.D., Lieutenant-Colonels A. R. Colwell and W. C. B. Harvey, Majors H. M. Cutler, J. Byrne, D. de la F. Henry, T. E. Y. Holcombe, H. McL. Hollingworth, M.C., and J. Z. Huie, Captain (Honorary Major) F. S. Hansman, Captains A. A. Callen, G. Archbold, G. H. H. Gall, S. G. Iceton, F. M. C. Jones, F. A. E. Lawes, J. H. W. Leadley, R. B. Lynch and H. H. O'Connor.

ROYAL AUSTRALIAN AIR FORCE.

Permanent Air Force: Medical Branch.

Walter James Slaven (052749) is appointed to a short-service commission, on probation for a period of twelve months, 22nd September, 1952, with the rank of Flying Officer.

The following officers are appointed to permanent commissions, 23rd September, 1952: Group Captain R. B. Davis (033026), Wing Commander G. C. V. Thompson (033034), Squadron Leaders J. B. Craig (05799), L. R. Trudinger (033059), G. A. Leyland (04397), M. C. Clarke (011321) (Acting Wing Commander).

Active Citizen Air Force: Medical Branch.

No. 22 (*City of Sydney*) *Squadron.*—Arthur Wilmot Raymond (025592) is appointed to a commission, 16th August, 1952, with the rank of Flight Lieutenant.

DISEASES NOTIFIED IN EACH STATE AND TERRITORY OF AUSTRALIA FOR THE WEEK ENDED DECEMBER 13, 1952.[1]

Disease.	New South Wales.	Victoria.	Queensland.	South Australia.	Western Australia.	Tasmania.	Northern Territory.[2]	Australian Capital Territory.	Australia.[3]
Acute Rheumatism	3(1)	1	..	..	..	..	..	..	4
Amœbiasis	..	2(1)	..	..	..	..	..	..	2
Ancylostomiasis	..	..	..	..	..	..	..	..	..
Anthrax	..	..	..	..	..	..	..	..	..
Bilharziasis	..	..	..	..	..	..	..	..	..
Brucellosis	..	..	..	..	..	..	..	..	..
Cholera	..	..	..	..	..	..	..	..	..
Chorea (St. Vitus)	..	..	..	..	..	..	..	..	..
Dengue	..	..	..	..	..	..	..	..	..
Diarrhœa (Infantile)	2(2)	..	8(5)	..	..	..	..	..	10
Diphtheria	6(5)	1(1)	4(2)	..	3(3)	..	..	..	14
Dysentery (Bacillary)	..	2(2)	1(1)	2(2)	2(2)	..	..	2	9
Encephalitis	..	..	..	..	..	..	..	..	..
Filariasis	..	..	..	..	..	..	..	..	..
Homologous Serum Jaundice	..	..	..	..	..	..	..	..	..
Hydatid	..	..	..	..	..	..	..	..	..
Infective Hepatitis	..	5	..	..	19(11)	..	..	..	24
Lead Poisoning	..	..	..	..	..	..	..	..	..
Leprosy	..	..	..	..	1	..	..	..	1
Leptospirosis	..	..	..	..	..	..	..	..	..
Malaria	..	..	..	..	..	..	..	..	..
Meningococcal Infection	2(1)	2(1)	..	..	..	..	..	..	4
Ophthalmia	..	..	..	..	..	..	..	..	..
Ornithosis	..	..	..	..	..	..	..	..	..
Paratyphoid	..	..	..	..	..	..	..	..	..
Plague	..	..	..	..	..	..	..	..	..
Poliomyelitis	18(10)	18(9)	6(2)	8(3)	2(1)	12(3)	..	..	64
Puerperal Fever	..	..	1(1)	..	..	..	..	..	1
Rubella	..	95(55)	1(1)	..	15(3)	..	..	..	111
Salmonella Infection	..	..	..	..	2(1)	..	..	..	2
Scarlet Fever	14(5)	17(14)	6(5)	5(5)	4(1)	..	..	..	46
Smallpox	..	..	..	..	..	..	..	..	..
Tetanus	..	..	1	..	2(2)	..	..	..	3
Trachoma	..	..	..	..	..	..	..	..	..
Trichinosis	..	..	..	..	..	..	..	..	..
Tuberculosis	49(40)	12(8)	32(15)	17(11)	13(9)	4(1)	..	..	127
Typhoid Fever	..	2(2)	..	..	1(1)	..	..	..	3
Typhus (Flea-, Mite- and Tick-borne)	..	..	1	..	..	..	..	..	1
Typhus (Louse-borne)	..	..	..	..	..	..	..	..	..
Yellow Fever	..	..	..	..	..	..	..	..	..

[1] Figures in parentheses are those for the metropolitan area.

[2] Figures not available.

[3] Figures incomplete owing to absence of returns from the Northern Territory.

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Honours.

THE following promotions and admissions have been announced in the Venerable Order of the Hospital of Saint John of Jerusalem.

Dr. C. A. Verco, Major-General F. A. Maguire and Lieutenant E. A. H. Russell have been promoted to the grade of Knight.

Dr. Katie Ardill Brice has been promoted to the grade of Dame.

Dr. R. S. Steel, Dr. H. H. Hurst, Dr. P. C. James and Brigadier C. W. Nye have been promoted to the grade of Officer.

Dr. C. D. Bateman, Dr. R. Swinburn and Dr. N. J. Caldwell have been admitted in the grade of Serving Brother.

Deaths.

THE following death has been announced:

WAINE.—John Grenville Waine, on January 7, 1953, at Narrandera, New South Wales.

Australian Medical Board Proceedings.

QUEENSLAND.

THE following have been registered, pursuant to the provisions of *The Medical Acts*, 1939-1948, as duly qualified medical practitioners.

Allison-Levick, Jack, M.B., B.S., 1952 (Univ. Queensland); Barnes, Keith Reginald, M.B., B.S., 1952 (Univ. Queensland); Bell, John, M.B., B.S., 1952 (Univ. Queensland); Biggs, Arthur Bruce, M.B., B.S., 1952 (Univ. Queensland); Borzi, Guiseppe, M.B., B.S., 1952 (Univ. Queensland); Bottomley, William Parker, M.B., B.S., 1952 (Univ. Queensland); Bradfield, Geoffrey Frank, M.B., B.S., 1952 (Univ. Queensland); Brand, Phyllis Lynette, M.B., B.S., 1952 (Univ. Queensland); Brodie, Magdalene, M.B., B.S., 1952 (Univ. Queensland); Brown, Campbell Villiers, M.B., B.S., 1952 (Univ. Queensland); Busch, Colin Reginald, M.B., B.S., 1952 (Univ. Queensland); Cameron, John Henry, M.B., B.S., 1952 (Univ. Queensland); Carroll, Gavan John, M.B., B.S., 1952 (Univ. Queensland); Denning, Neville Owen, M.B., B.S., 1952 (Univ. Queensland); Drake, Richard Francis, M.B., B.S., 1952 (Univ. Queensland); Eckert, John Paul, M.B., B.S., 1952 (Univ. Queensland); Edye, Mary Rae, M.B., B.S., 1952 (Univ. Queensland); Emmerson, Bryan Thomas, M.B., B.S., 1952 (Univ. Queensland); Ferguson, Ian Ross, M.B., B.S., 1952 (Univ. Queensland); Feltoe, John Francis, M.B., B.S., 1952 (Univ. Queensland); Ferrier, Melville Chaloner, M.B., B.S., 1952 (Univ. Queensland); Foley, James Peter, M.B., B.S., 1952 (Univ. Queensland); Foxton, Harold Alexander Bell, M.B., B.S., 1952 (Univ. Queensland); Fulcher, Barrie Howes, M.B., B.S., 1952 (Univ. Queensland); Gallagher, Colin Gordon, M.B., B.S., 1952 (Univ. Queensland); Glover, Noel Stanley, M.B., B.S., 1952 (Univ. Queensland); Gooch, Richard Neil, M.B., B.S., 1952 (Univ. Queensland); Goodwin, Robert Molesworth, M.B., B.S., 1952 (Univ. Queensland); Gregg, Ronald Francis, M.B., B.S., 1952 (Univ. Queensland); Hamilton, William James, M.B., B.S., 1952 (Univ. Queensland); Haughton, Charles Steele, M.B., B.S., 1952 (Univ. Queensland); Hirschfeld, Brian Eugen Eliott, M.B., B.S., 1952 (Univ. Queensland); Hocker, Geoffrey Arthur, M.B., B.S., 1952 (Univ. Queensland); Hyslop, Graham McLeod, M.B., B.S., 1952 (Univ. Queensland); Ilett, Geoffrey Byford, M.B., B.S., 1952 (Univ. Queensland); Macleod, Roy Maxwell, M.B., B.S., 1952 (Univ. Queensland); Mactaggart, Peter, M.B., B.S., 1952 (Univ. Queensland); Mitchell, Peter Gray, M.B., B.S., 1952 (Univ. Queensland); Moy, Herbert Henry, M.B., B.S., 1952 (Univ. Queensland); Murdoch, Malcolm Herbert Roche, M.B., B.S., 1952 (Univ. Queensland); Murphy, Kevin James, M.B., B.S., 1952 (Univ. Queensland); McCabe, William Joseph, M.B., B.S., 1952 (Univ. Queensland); O'Connor, Brian Thomas, M.B., B.S., 1952 (Univ. Queensland); O'Rourke, Desmond Anthony, M.B., B.S., 1952 (Univ. Queensland).

Nominations and Elections.

THE undermentioned have applied for election as members of the New South Wales Branch of the British Medical Association:

Bassett, Ray, M.B., B.S., 1952 (Univ. Sydney), Singleton District Hospital, Singleton, New South Wales.

McDonald, Colin James, M.B., B.S., 1951 (Univ. Sydney), 3 Pindari Avenue, Mosman, New South Wales.

Walsh, Desmond Michael, M.B., B.S., 1951 (Univ. Sydney), 40 National Park Street, Hamilton, New South Wales.

Traub, Erich, provisionally registered in accordance with the *Medical Practitioners Act*, 1938-1950, Section 17 (1) (c), 2 New South Wales Head Road, Edgecliff, New South Wales.

Diary for the Month.

JAN. 20.—New South Wales Branch, B.M.A.: Medical Politics Committee.
JAN. 22.—Victorian Branch, B.M.A.: Executive Meeting.
JAN. 28.—Victorian Branch, B.M.A.: Council Meeting.
JAN. 30.—Queensland Branch, B.M.A.: Council Meeting.

Medical Appointments: Important Notice.

MEDICAL PRACTITIONERS are requested not to apply for any appointment mentioned below without having first communicated with the Honorary Secretary of the Branch concerned, or with the Medical Secretary of the British Medical Association, Tavistock Square, London, W.C.1.

New South Wales Branch (Medical Secretary, 135 Macquarie Street, Sydney): All contract practice appointments in New South Wales.

Victorian Branch (Honorary Secretary, Medical Society Hall, East Melbourne): Associated Medical Services Limited; all Institutes or Medical Dispensaries; Australian Prudential Association, Proprietary, Limited; Federal Mutual Medical Benefit Society; Mutual National Provident Club; National Provident Association; Hospital or other appointments outside Victoria.

Queensland Branch (Honorary Secretary, B.M.A. House, 225 Wickham Terrace, Brisbane, B17): Brisbane Associated Friendly Societies' Medical Institute; Bundaberg Medical Institute. Members accepting LODGE appointments and those desiring to accept appointments to any COUNTRY HOSPITAL or position outside Australia are advised, in their own interests, to submit a copy of their Agreement to the Council before signing.

South Australian Branch (Honorary Secretary, 178 North Terrace, Adelaide): All Contract Practice appointments in South Australia.

Western Australian Branch (Honorary Secretary, 205 Saint George's Terrace, Perth): Norseman Hospital: all Contract Practice appointments in Western Australia. All government appointments with the exception of those of the Department of Public Health.

Editorial Notices.

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